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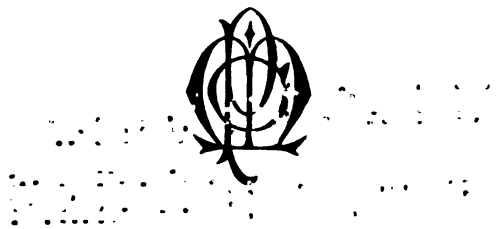

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INJURIES TO THE SEMILUNAR CARTILAGES OF THE KNEE-JOINT.*

By RUTHERFORD MORISON, F.R.C.S.,

Professor of Surgery, University of Durham.

LADIES AND GENTLEMEN,—Injuries to the semilunar cartilages of the knee-joint are common in miners, so that in Newcastle Infirmary the surgeons have exceptional opportunities for seeing them. So lately as on October 8th of last year, one of my colleagues, Mr. Martin, read an interesting and instructive paper in London before the Surgical Section of the Royal Society of Medicine, which has attracted special attention to this important subject.

Until recent years the pathology of the conditions in the knee-joint which caused sudden, sometimes serious, always troublesome, and often fugitive symptoms, were ill understood. Clinically they were grouped together under the title of "Hey's internal derangement of the knee-joint." A definite pathological diagnosis is now possible in the majority of these cases, and the treatment of them has consequently become reasonable. The saying, "It is sometimes impossible to make an accurate diagnosis, but it is always a mighty good thing to try," emanating from Dr. W. Mayo, of America, is one to be remembered.

There are still some unsettled points for discussion which are of considerable importance, and it is chiefly in the hope of interesting you, and also because I have recently had some unusual experiences in these, that I have taken up the consideration of this subject to-day.

The first concerns the most favourable position of the limb to allow of the injury. Two distinct and opposite views were expressed in the London

* Two clinical lectures delivered in the Royal Victoria Infirmary, Newcastle-on-Tyne.

discussion, Mr. Martin expressing the opinion, based on his large experience, that it nearly always occurred during flexion, Mr. Walton, who had made careful experiments on dead bodies, believing that it could only occur during extreme extension. The overwhelming majority of the cases here occur with the limb in a condition of flexion, generally acute, and added to this a twist usually with adduction at the knee. In some, however, it occurs at the end of forced extension, and the following account given by an exceptionally intelligent patient is a case in point.

CASE 1.—I. L—, æt. 30 years, was admitted to the Royal Victoria Infirmary on October 17th, 1912.

History of accident.—Eleven months previous to admission the patient was playing football. While the ball was in the air he kicked at it, and *at the moment his knee was fully and forcibly extended* he felt a pain so acute that he fell down.

On getting up he found his knee "locked," and next day it was much swollen.

Since then he has had frequent recurrences of the pain and locking, brought about by small provocation, and has been unable to work.

Physical signs.—There was some wasting of the muscles of the thigh and leg, and a tender spot over the anterior and inner side of the knee-joint.

Operation (Mr. Morison) October 19th, 1912.—The greater part of the cartilage was detached circumferentially, and the posterior end of the fragment, which was split transversely, was lying in the joint. A second small split was found on the inner margin of the portion projecting into the joint.

The second point to which I wish to draw your attention concerns the nature of the injury. It is essential, if treatment is to be carried out on rational lines, that clear views should be held as to this, and the success or failure of operative treatment *may* depend entirely upon a diagnosis of the exact nature and the position of the damage done.

In the 'Lancet' of February 27th, 1909, I drew attention, by a paper entitled "Injuries to the Semilunar Cartilages of the Knee," to what I believed to be a fact—that in every case with a certain definite history rupture of the cartilage had occurred. Previous to this a description of the conditions by which similar symptoms could be produced encumbered the brains of surgeons and still fills pages of the text-books, but gradually in America as well as in this country the clouds are clearing, and the view that fracture of the cartilage with displacement of the broken portion is the cause of the joint derangement is steadily gaining ground.

The immediate history, to which I attach so much importance, then as now, is one consisting of three sequential events.

(1) A knee injury followed by such severe pain as often to make the patient faint and fall, or turn him sick.

(2) Locking of the joint, *i. e.* inability to fully extend it, and a feeling of painful tension on the hamstrings when attempting to do so, whilst the power to flex it is retained.

(3) Swelling of the joint in the course of a few hours after the injury.

A confirmatory symptom might be a crack heard at the time of the accident, and this has several times been described to me, possibly in exaggerated terms, as "like a pistol going off," but it is not constantly heard, and may mean little if it is, for many joints crack on occasion.

The after-history concerns two points, and to the first I may say that I attach, as the patient is likely to do also, the very greatest diagnostic importance. It is recurrence of the condition from time to time after recovery.

The second is that the intervals are free from any disturbance whatever. If they are not a diagnosis of fracture is either wrong or there are complications.

CASE 2.—L. W—, æt. 53 years, miner. Admitted October 28th, 1912.

A year's history of pain in right knee, with limping on walking, and aching at night. Knee worse during wet weather.

Three weeks ago while in the pit his leg caught on a stone and his knee twisted under him. He was unable to straighten his knee, but continued his shift (five hours) and walked home with difficulty. His knee swelled up.

Since accident has not walked at all.

Signs.—Marked osteo-arthritic creaking in joint. Tender spot and wasting of muscles.

Operation (Mr. Morison), October 29th, 1912.—Hypertrophied alar ligament and cartilaginous fringe removed. Also anterior third of internal cartilage, the latter with two definite cracks.

What is the cause of all this disturbance?

After what I have said you may expect the answer to be—the fracture; but that is surely wrong. Cartilage, unlike bone, if it has any nerves at all, has a very poor supply, and sensation in it is certainly of a very low quality. The pain is the result of stretching of the ligaments, either due to locking of the joint, by displacement of the fractured fragment, or to the original injury. It is important to note this, for in exceptional cases no severe pain, such as we regard as of great diagnostic importance, may be felt at the time of the injury, and the patient may almost have forgotten the accident when a typical attack follows some trifling jerk or twist of the leg, especially such as would occur in catching the big toe and front and inner part of the foot against any obstacle.

Our notes of cases relate how the painful locking sometimes occurred for the first time after turning over in bed, and it is obvious that an injury so trifling could not have caused the rupture of the cartilage found at operation. The explanation I offer of this is, that fracture occurred with the first injury, and that displacement of the separated portion between the bones did not happen then, but later, when the chief damage apparently resulted from a wholly inadequate force. With the displacement painful stretching of the ligaments is brought about by the mechanical obstacle interposed between the opposed surfaces of the femur and tibia.

Now the law of this country provides that any workman injured in the course of his employment can claim compensation for the disability from which he suffers as a consequence, and some of the best class of workmen, those most eager for work, may resume their occupation at once, or in a short time after the fracture has occurred, and become disabled at a later date by the displacement of the ruptured fragment. A grave injustice to the man may, and has, followed ignorance of this point on the part of a doctor.

Locking.—A history of this may be all the knowledge obtainable of it (a symptom), or the patient may come with the knee "locked" (a sign). By the term "locking" all of us have thought that a condition which prevents full extension of the joint, but allows of free flexion, is meant, and with very rare exceptions this would be the correct interpretation. It may, however, be of the greatest importance to recognise that in exceptional cases locking occurs during flexion, while extension is free, or that locking may occur at one time during flexion and on another occasion during extension.

Case 3.—W. P—, æt. 47 years, miner; admitted November 25th, 1912.

Sixteen years' history. While filling a tub, with his knee slightly flexed, his foot slipped and his knee bent sharply inwards with a crack, causing severe pain. He did not fall or cry out but continued his work, and then limped home (quarter of a mile), with pain over inner side of his knee. His knee swelled up. He worked for a week and then had to lie off a fortnight. His knee was locked when in an extended position.

He has had repeated attacks since, followed by *locking in both extended and flexed positions.*

Signs. — Wasting; pain on internal rotation, otherwise movements normal; tender spot on inner and front part of joint.

Diagnosis.—Rupture of internal cartilage, with fragment projecting about middle of joint.

Operation (Mr. Morison), November 26th, 1912, —Anterior two thirds of internal semilunar cartilage removed. *Pathology:* Double split of cartilage, a tag projecting into the joint.

The importance of this fact and of its bearing upon diagnosis will be best emphasised by the relation of another case, *because if an accurate diagnosis had not been made the joint would certainly have been closed, or some useless tinkering done,* and the patient would have been unrelieved.

Case 4.—J. R—, æt. 41 years, miner; admitted November 18th, 1912.

Twelve weeks before while lifting a tub his knee twisted outwards; it was fully extended and his heel slid inwards. He heard a click, had severe pain over the internal cartilage, and he dropped. After a short time he limped home supported by two men. His knee never locked until he got to bed, when it locked while fully extended. He got someone to move it and it locked again, temporarily, in a flexed position. His knee became swollen. Locking in the extended position had happened three times since.

Signs.—Slight muscular wasting, no impairment of movement. Tender spot over antero-internal aspect of joint.

Diagnosis. — Posterior rupture of internal cartilage.

Operation (Mr. Morison), November 19th, 1912.

—On opening the joint both semilunar cartilages appeared to be normal. The rupture was only seen with difficulty, *after acute flexion of the joint, and after the anterior half of the cartilage had been separated circumferentially, and drawn forcibly forwards.*

Swelling of joint.—It seems difficult to give an entirely satisfactory reason for the rapid effusion into the synovial cavity which follows this accident.

The usual explanation offered is that it is due to "traumatic synovitis," and this is probably as nearly correct as we can get it. Inflammation, in my systematic course, is described as of two varieties: (1) Physiological, protective and wholly constructive; and (2) pathological, commencing when stasis develops, always locally more or less destructive, whatever may be its effects on the body as a whole. This "traumatic" synovitis is wholly protective.

Most of you will remember as boys, and perhaps as girls too, "melting snails with salt." The same reaction as you saw there is a common protective response to irritation, and it occurs to a marked degree in all serous cavities such as pleura, peritoneum and joints, in all of which fluid effusion quickly follows any traumatism of the lining membrane.

The crack which may be heard at the time of the accident we assume to be due to the fracture of the cartilage which has taken place.

Recurrence of the symptoms.—The cause of this is *non-union of the fracture*, and is the result of sudden displacement of the loose fragment between the ends of the bones.

You will note that a tender spot over the anterior and inner portion of the joint and wasting of the muscles is mentioned in most of the case reports. These signs are not special to this condition, but are found in all organic disturbances of the knee-joint, so that no special diagnostic importance attaches to them.

With some experience the majority of patients learn how to replace the broken portion, and if you wish to retain their confidence you will ask them to try the effect of their own manipulations before you risk your reputation by making any attempt on your own account. Some of them succeed regularly by methods which no one can understand or appreciate, and it would be useless to attempt to describe them. Ordinarily, if firm flexion followed by steady extension fails, no further effort should be made, and the limb is to be left in the most comfortable position, supported by sand-bags with some hot application over the joint. As a rule, then, in the course of a few days some small spontaneous movement produces a sudden change and the joint has righted itself again. As soon as the displacement is rectified the limb should be put in effective splints and kept there from six to eight weeks. For some months after the injury special care should be taken of it, the object being to secure union of the broken cartilage.

After recurrence the proper course is to recommend operation.

There is another displacement to which I wish to draw your special attention because a disagreeable experience, from which I wish you to derive the benefit I have gained myself, has taught me the importance of it. I trust after hearing this that you will not believe, as all of us occasionally are apt to do, that these details are of "mere academic interest," which generally means to say, at the least, lack of enthusiasm. Attention to them may, and does, make the difference between success and failure.

The displacement I allude to is one we describe as the bucket-handle variety, and the name almost explains the condition. It is due to a circumferential tear with separation of the torn part, except anteriorly and posteriorly, and displacement of the torn portion outwards as a loop into the joint. This is not a usual fracture and displacement, though it is not extremely rare, because I have operated upon four such cases during two recent months.

Case 5.—S. G—, æt. 24 years, miner; admitted December 12th, 1912.

History.—Six months ago, while hewing, he slipped and twisted his knee inwards, his knee being bent at the time. He felt something like a crack and had sudden pain which made him fall down. At the time he was unable to straighten his leg and had to lift himself up by holding on to a tub. He limped home with great pain and his knee swelled up. He laid up three weeks and a week later his knee gave way with similar symptoms. Two attacks since. Pain in joint on exercise.

Signs.—No wasting. Tender spot and pain on internal rotation.

Diagnosis.—Ruptured internal cartilage.

Operation (Mr. Morison), December 21st, 1912.—Open removal. *Pathology:* Bucket-handle rupture.

CASE 6.—J. F—, æt. 34 years, deputy; admitted January 6th, 1913.

History.—Five weeks ago, while working in the pit in a three-foot seam with his leg crossed under him, a stone fell on to his back and forced him over, putting all his weight on to his left leg. He felt a click in his knee and had severe pain and shouted out. He lay still for a few minutes, and in getting up his knee "went in" again with a click. His knee swelled that night. Since the first attack he has had about twenty, his knee locking in a slightly flexed position, and it has to be manipulated in again. A fortnight ago he was unable to unlock his knee for nearly a day, and he walked home with his knee slightly flexed and very painful.

Signs.—Wasting of muscles; tenderness. Flexion limited, but can straighten the joint.

Diagnosis.—Ruptured internal cartilage.

Operation (Mr. Morison), January 7th, 1913.—Open removal. *Pathology:* Bucket-handle rupture.

CASE 7.—G. M—, æt. 41 years, miner; admitted November 11th, 1912.

History.—Twenty-six weeks ago, while hewing, his left knee slipped inwards, his knee being flexed at the time. No crack, but bad pain; he did not fall and had no locking. On walking had a peculiar grating in the joint. He worked for six days afterwards. When he put any weight on the joint he felt something "slipping" on the inner side. The knee became swollen the day after accident, and had remained swollen. Flexion limited. Joint stiff and movements are painful after a resting period.

Signs.—Muscular wasting and inflammation in joint. Tender spot; marked creaking; some limping; full flexion hurts him; some grating in other knee-joint.

Operation, November 14th, 1912.—Anterior three quarters of internal cartilage removed. *Pathology:* "Bucket-handle," with a second split near anterior end. Some free fluid in joint and signs of osteoarthritis.

CASE 8.—A. C—, æt. 22 years, fireman; admitted November 5th, 1912.

History.—Four months ago his knee was twisted inwards while at work, and he fell to the ground in great pain and with inability to straighten the limb. Got home with difficulty and was laid up for some days. Knee much swollen. Since then frequent locking of joint, relieved by flexion and extension.

Operation, November 9th, 1912.—Internal cartilage found split down its middle without detachment of either end—bucket-handle variety—and projecting into joint. Cartilage removed.

As a rule with a free incision and good exposure of the joint the displaced loop at once comes into view and no mistake is possible, but every now and again the loop lies in the intercondyloid notch and hugs the internal condyle so closely that it requires careful searching for, and there may be a little difficulty in pulling it out of its seeming hiding-place. The frayed edge of the still normally attached cartilage under such circumstances is possibly removed in the belief that the explanation of the symptoms has been found, but this belief is rudely shaken when the patient returns "no better," and you will be lucky if he comes back to you.

The disagreeable experience which taught me most of this concerns a patient on whom I operated in the Royal Victoria Infirmary. He had a history and symptoms so typical that it was impossible to misunderstand them and I opened his knee-joint. The notes say that I removed "the anterior portion of the cartilage, which was frayed at its edge." Four months later he returned with the same symptoms as before operation. Mr. Richardson again opened his joint and "removed the remaining posterior portion of the damaged cartilage." Six months later he came back "no better," and unable to work because of frequent painful locking of his knee. For the third time I opened his joint more freely than before and discovered a loop of cartilage hugging his internal femoral condyle, and still retaining an anterior and posterior attachment to the remaining extremities of the internal semilunar cartilage. Ever since I have had "a good look round," especially in cases where sufficient has not been found to account for the whole trouble, and I have so often discovered what I was looking for that I am convinced many of the patients who are "no better" after operation, or "who have osteoarthritis" after it, or in whom "adhesions are the cause of all the trouble" of which they still complain, are the victims of a mistaken diagnosis, supported by operation through a keyhole incision.

The operation and incision I still employ are fully described in the article previously referred to ('Lancet,' February 27th, 1909), so that I need not occupy your time in useless recapitulation, but I would like to record a recent case showing that from the patient's point of view the operation is not *always* satisfactory, though curiously enough it is one of the most successful in surgery. My expression of surprise at its success arises from a strong belief that no operation which interferes so materially with the anatomy and physiology of the body can be done with impunity, though it must be admitted that in these cases any complaint of failure is rare.

CASE 9.—E. S—, æt. 20 years, miner; admitted July 2nd, 1912.

History.—While playing football fifteen weeks ago, he was about to shoot when someone charged him, his knee being flexed and "knocked in over." He heard a crack and felt sudden severe pain, fell to the ground and was carried off. He was unable to straighten his knee and next morning it was swollen. He laid up for a month. On getting up his knee locked again once or twice and he was kept off work for six weeks. Since then he had worked in the pit and his knee had locked about thirty times. He is now able to manipulate it himself and straighten it with a click when it locks.

Signs.—Knee is swollen, and a tender spot over anterior and inner part of joint.

Diagnosis.—Ruptured internal cartilage.

Operation (Mr. Morison), July 4th, 1912.—The left internal cartilage was found ruptured longitudinally in its anterior portion and was removed.

Re-admitted November 8th, 1912.

Subsequent history.—Patient was at full work six weeks after the operation. He remained quite well until two days before admission, when in coming downstairs he slipped, and before he knew where he was he found himself sitting on his doubled-up left leg. He had very severe pain in the left knee-joint and could not straighten it.

The joint swelled up at once.

Signs.—On admission, joint swollen, tense and very painful. All the signs of inflammation present. Joint held in a position short of full extension. No lateral mobility.

Mr. Morison thought that possibly the position of the internal cartilage remaining behind had been ruptured.

Operation, November 12th, 1912.—Joint tensely distended by deeply blood-stained synovial fluid, which on incision escaped with great force. The synovial membrane was swollen and bright red. The anterior crucial ligament was partially ruptured. A strand of fibres the size of a chrysanthemum petal had been detached inferiorly and the blood-staining was due to clot on this, showing where the bleeding had come from. The torn portion of ligament was excised. The remnant of internal cartilage and the rest of the joint were normal. The inferior aspect of the cartilage of the internal femoral condyle was roughened as if from early osteoarthritic changes. The joint was emptied and completely closed and the leg was placed in lateral splints to keep it at complete rest. Uninterrupted recovery.

Two points about this case are noteworthy:

(1) That the injury was followed by signs of more active inflammation than is usual in these cases. There was severe pain, marked heat, tense swelling of the joint, and striking redness of the synovial membrane when the joint was opened. Locally the signs were those of an early acute septic arthritis, but the absence of serious general disturbance and the moderate rise in temperature present enabled us to exclude sepsis. What was the cause of the disturbance? You will remember that blood was found in considerable quantity in the joint, and experience has taught us that blood in any of the serous cavities causes inflammation so active as to be often followed by sufficient destruction to result in adhesions (fibrosis).

(2) The second point concerns the injury and the change found since the previous operation on the inner condyle of the femur. Is it possible that this serious injury, rupture of the crucial ligament and the so-called osteoarthritic change, were both due to the removal of the elastic pad called the internal semilunar cartilage? I suspect that both were due to this.

I want to emphasise these facts because I have learned them since the publication of my last paper. *First*, that rupture of the cartilage can occur without the overwhelming pain we have been accustomed to expect, and that the pain is due to *displacement of the fragment* between the bones and not to the fracture. *Second*, that although "locking" is rare except in extension (Fig. 1), yet if the fracture is posterior and the displaced

fragment gets between the bones behind, the "locking" will occur during flexion (Fig. 3). A "locking" that occurs either during extension or of rupture—the bucket-handle variety (Fig. 4)—the displaced fragment may be overlooked during an operation, unless the possibility of such an

Internal semilunar cartilage of right side to illustrate manner of "locking" in varieties of rupture (semi-diagrammatic). A. Anterior. P. Posterior attachments.

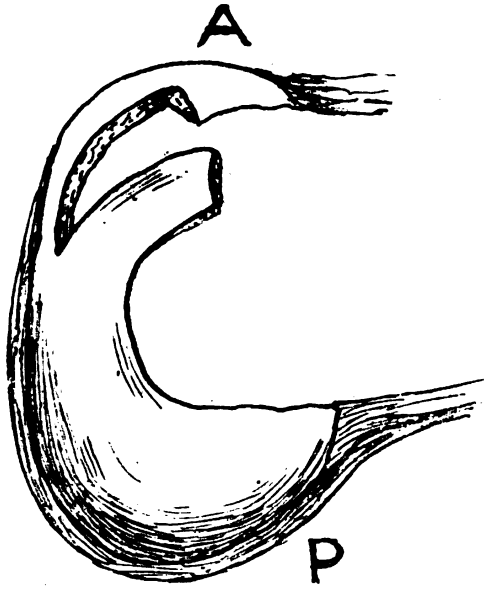

FIG. 1.—The knee cannot be fully extended.

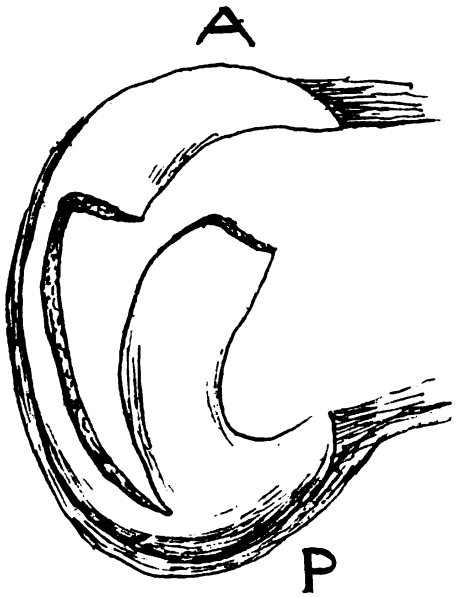

FIG. 2.—Full flexion or extension may be interfered with.

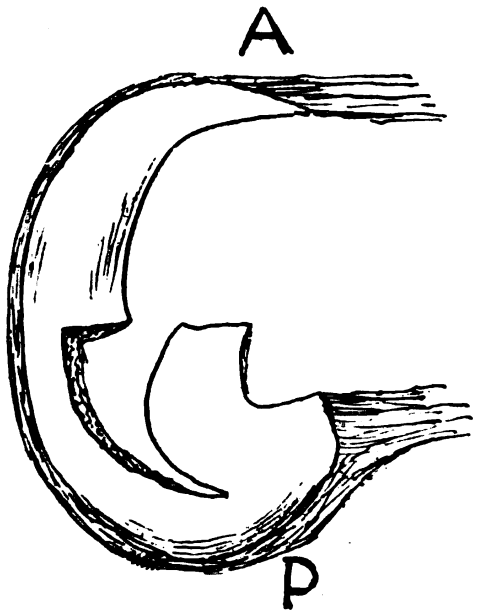

FIG. 3.—The knee cannot be fully flexed.

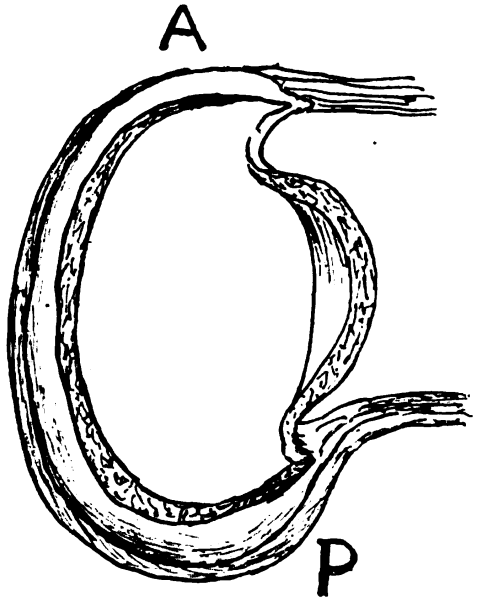

FIG. 4.—Bucket-handle variety of rupture.

flexion, sometimes the one and sometimes the other, is due to a pedunculated fragment long enough to reach either the anterior or posterior part of the joint (Fig. 2). *Third*, that in one form occurrence is remembered and a careful search made.

April 9th, 1913.

TREATMENT BY MASSAGE AND MOVEMENT, PARTICULARLY IN RELATION TO FRACTURES.*

By R. C. ELMSLIE, M.S., F.R.C.S.,
Assistant Surgeon to the Hospital, and Orthopædic Surgeon to St. Bartholomew's Hospital.

MASSAGE consists in treatment of some part of the body by friction, pressure or kneading. In ordinary massage these manipulations are applied by the hand in various ways. But similar effects can be obtained by applying the same methods by means of some mechanical contrivance, as in certain forms of Zander apparatus, or by an electrically driven machine. Apart from local effects on the skin, the effects of massage may be considered as they affect the circulation, local and general, and as they affect the deeper tissues. The effect upon the circulation is largely mechanical. Centripetal stroking tends mechanically to increase the flow of blood in the veins and of lymph towards the trunk, and so by emptying the veins to increase the rate of flow in the arteries. But in addition to this it soon dilates the vessels and produces a local hyperæmia.

The effects upon the deeper tissues are partly circulatory, partly mechanical. The hyperæmia extends to the deeper tissues, particularly if the friction is applied deeply, or if it is accompanied by kneading. This deep hyperæmia, by assisting the removal of waste products and by bringing nutriment, rapidly restores a tired muscle to its normal state. But further, if any local inflammation is present, it assists in the local fight against an infecting agent, promotes the absorption of inflammatory products, and if any new fibrous tissue has been formed, by vascularising it, assists in softening and stretching it, and even in procuring its absorption.

The actual mechanical action of kneading also assists in stretching any such inflammatory fibrous tissue.

One other local effect of massage must be alluded to. The French school of masseurs have shown that light stroking or friction applied regularly along the same lines over a limb produces after a few minutes an actual local anæsthesia, which affects not only the surface but also the deeper parts, so that pain is relieved, and muscular spasm, due to pain, may even be abolished.

To many masseurs, specially those trained in the strict Swedish school, the idea that massage can be painless seems absurd, and an anæsthetic effect a frank impossibility. But this simply means that these workers have not realised the value of this painless treatment. The popularisation of this form of massage, specially in the treatment of fractures, is due to Championniere. This surgeon's definition of the sort of massage to be applied to fractures is a gentle centripetal friction, slowly and regularly repeated, stroking as you would stroke a cat, "little more than a caress." Unless this idea of painless massage is realised as a preliminary to the treatment of fractures by this method, difficulties and disappointments will be encountered.

Movement in treatment means the carrying out of normal joint movements within their normal limits. It seems hardly necessary to point out that when we speak of moving cases of fracture, we mean these joint movements and not movements of the ends of the fractured bone on each other, yet one sometimes finds critics discussing the treatment as if this was what was meant.

Movements are of two sorts. Active movements are carried out by the patient himself; they will of course strengthen the muscles used, but are not likely to increase greatly the total range of movement. Passive movements are carried out by the manipulator. They may be gentle or forcible. Active movements may be simple, or may be resisted either by machinery or by pressure applied by the manipulator, so as to increase the work done by the muscles. Further, active movements may be supplemented by passive, the active movement being carried out to its full extent and an additional passive stretching carried out by the manipulator at the end.

All these varieties of movement can be carried out by machinery of the Zander or Pendel type. Such machinery has the advantage of reducing the labour of the manipulator, but practically everything that can be done by such machinery can also be done by hand, and done with more accuracy and delicacy.

Movements may be carried out with the idea of preserving the function of a joint, or of improving

* Read at a meeting of the Æsculapian Society.

the function of a damaged joint by restoring its proper range of movement, or with the idea of improving the strength or activity of the muscles. The sort of movement to be employed and the method of carrying it out must depend upon the end desired. And this end should be borne in mind during the treatment.

In order to illustrate practically the uses of massage and movement I propose to take (1) sprains, (2) acute arthritis, and (3) fractures.

Sprains.—A sprain may be defined as a contusion in the neighbourhood of a joint. Pathologically most sprains consist in the tearing of some structure, most often of a ligament. This tearing is followed within a short time by an effusion into or around the joint, first of blood from ruptured vessels, later of exudate from the vessels.

The old-fashioned method of treatment in such cases was to rest the part and apply cold lotions to lessen the effusion, then to rest the joint until the effusion had disappeared, and then to attempt to restore movement. Apart from the waste of time involved this method had another serious disadvantage. An effusion, whether of blood or of plasma, tends, if left, to undergo organisation, fibrous tissue being formed; this results in the production of intra-articular and peri-articular adhesions, which take a long time to stretch or absorb. Indeed, if such adhesions become dense, they may prove incapable of being stretched. A history in a case in which such adhesions arise may go thus.

A patient wrenches his knee, there is considerable effusion, and for this the joint is rested for a month, no movement being allowed. At the end of this time the effusion is gone, but the joint is stiff and the muscles feeble. Movement is then started, but adhesions have formed which are stretched by any but small movements, so that directly any considerable movement is carried out an adhesion is stretched, the joint becomes painful, and the stretching of the adhesion produces a fresh effusion. This effusion is considered an indication for further rest, which in turn produces increased atrophy of the muscles, and allows the adhesions to consolidate again. A vicious circle is now reached, and unless the joint is properly treated, recovery may be indefinitely delayed. The proper treatment of such a joint is to move it fully under an anæsthetic, breaking down all adhesions, to prevent immediate effusion by the method to which I shall refer presently, and to start moving the joint the next day.

To come to a more rational method of treatment. The evident indications are to limit the effusion, to promote its rapid absorption, and to restore the mobility of the joint before any firm adhesions have had time to form. The limitation of an effusion can be most easily carried out by the simple mechanical method of applying pressure. There are two particularly good methods of applying this, each of which has its advantages—by strapping, and by a compression bandage. The second is perhaps the most generally useful, and is specially indicated in those cases where, when a patient is first seen, there is already a good deal of effusion, or where a large effusion is feared. It is carried out thus:

The whole limb for a distance of two or three inches on either side of the joint is swathed in sheets of absorbent wool about an inch thick. A domette bandage is then firmly applied, as much force as is possible being used. A firm elastic compression is thus kept up over the joint and its neighbourhood, and at the same time a certain small amount of movement is allowed. This small amount of movement has a use to which I shall allude immediately. If such a bandage is applied immediately after an injury, when it is removed twenty-four hours later it will generally be found that the effusion is very slight or even absent. Moreover, if such a bandage is applied over a joint into which an effusion has already taken place, it will often be found that this has disappeared in twenty-four hours.

Strapping applied directly to the joint is indicated rather in cases in which there is very little effusion, and particularly in such a joint as the ankle, when there appears to be a risk of the joint falling over into an unnatural position if it is subjected to weight. It has the great disadvantage that its daily removal for massage and replacement is tiresome.

The second object, the rapid removal of the effusion, is effected by massage. In addition to the ordinary methods of massage carried out daily auto-massage may be utilised. If a joint, such as the knee, is covered by a firm compression bandage, it can, in most cases, be moved from the first. The movements are small, because the bandage will

restrict them, but they are sufficient to vary the pressure upon the different parts covered by the bandage. These variations in pressure keep up a certain amount of auto-massage, which hastens the absorption of the effusion.

In order to hasten the recovery of the function of the joint, movements should be carried out from the first. The question arises whether by doing this there is any risk of preventing union of an important ligament which has been ruptured. This must vary in individual cases, but it may be said at once that there is very seldom any such risk. In such a joint as the knee there is practically no risk at all; movements may be carried out passively and actively from the first. And within a few days the joint may be safely used for walking, provided always that the bandage is kept applied. The ankle is an example of a joint in which greater care must be taken, more particularly if some ligament upon the inner side has been ruptured. There will then be a considerable risk of weakening the joint and producing a bad degree of valgus or flat foot. It is in such a case that the advantages of strapping are shown. As soon as the effusion has been reduced, strapping can be applied in such a way as to limit movement in the dangerous direction, in this case movements of eversion and abduction. Movements can then be carried out actively and passively, and very soon weight can be borne.

Cases of simple strain treated by such methods of bandaging, massage and early movement recover quickly. Needless to say it is essential to make an accurate diagnosis in the first place. Not only must a fracture, even the chipping off of a small piece of bone, be excluded, but an accurate estimate of the amount of damage done to the joint should be arrived at at the first examination; this will give the best indication of the amount of support that the joint will require during its treatment.

Acute arthritis.—In acute arthritis, due to a definite infecting agent, such as the gonococcus, the pathological changes in the joint differ considerably from those occurring after an injury. In the first place the effusion contains the micro-organism and also dead leucocytes—it is, in fact, pus. Further, there is an infiltration of the joint surfaces which may result in the destruction of the cartilage and in the stretching and softening of ligaments, the joint being thereby much weakened. During the acute stage gentle massage may help to diminish slightly the effusion, but movement will certainly tend to increase all the pathological changes. That is to say, if movements are carried out, there will be more effusion, more destruction of cartilage, and more softening of ligaments, and the whole process will be continued for a longer time. Movements are often advocated in these cases from the first because of the great liability to subsequent stiffness. But as this stiffness is directly due to the pathological processes the first indication is evidently to stop the progress of these, and this is best effected by a period of rest, during which the infection itself is treated by vaccines or otherwise.

Massage and gentle movements are indicated as soon as the acute inflammatory symptoms have subsided, and the extent to which the movements can be carried are best indicated by saying that they should be just sufficient to be felt to stretch the parts, without causing any considerable pain. Just as in the treatment of sprains we have neglected the uses of movement, so in an arthritis such as that due to the gonococcus, we have pushed early movement to an extent that is eventually harmful.

The aim of treatment by movement in a case of arthritis after the acute stage is passed is to gradually increase the range of movement of the joint until the normal range is attained. The period for which it is continued varies very greatly, from weeks to a year or more. It is common, however, to find a masseur wasting his time in attempting to restore movement in a joint that has been completely disorganised, and in which the joint surfaces are probably fixed together by fibrous tissue. In a case in which no apparent progress is being made an examination under an anæsthetic is indicated. This examination serves two ends. In the first place it informs us whether there is any possibility of securing additional movement, and in the second place it serves to break down any localised adhesions. If under an anæsthetic movements are obtained only with great difficulty and are never free, it may be guessed that there is definite fibrous ankylosis. In such a case any movement subsequently obtained is likely to be painful, and it is usually better to abandon the effort and to leave the joint stiff. In coming to this decision attention must of course be paid to

the joint affected and to the importance of its movement, *e.g.* a completely stiff elbow is a great disability, a completely stiff knee a much less serious thing.

Fractures.—In the treatment of fractures it is necessary to insist again upon two points. First that massage of fractures should be almost or quite painless, and second that movement means movement of the joints and not of the ends of the bones upon each other. The first objects are the prevention of effusion and the reduction of displacement. Considerable effusion and the occurrence of fracture blisters constitute the most serious difficulties in the rational treatment of fractures. It is remarkable that after the operation of osteoclasis, as also after wrenching a foot with the Thomas wrench, there is never any considerable effusion, nor do blisters occur. Yet the violence used must often be as great as is the case in many fractures. I believe that this immunity depends upon the fact that after these operations the injured part is at once covered with a firm bandage and with plaster, applied upwards from the most distal part of the limb. The effusion is thus prevented mechanically. I believe that if we were able to treat all fractures by the immediate application of firm compression bandage, we should avoid the occurrence of many of these troublesome complications. It is essential to remember that in applying such a bandage it must be taken up from the most distal part of the limb. If after an osteoclasis the plaster bandage is applied in such a way that it does not extend down to the level of the toes, it is almost certain that the fore part of the foot will swell. And if on account of this swelling the lowest part of the plaster is cut away, the swelling will extend, until the whole plaster has to be removed.

The second point to be attended to in the treatment of a fracture is the reduction of the displacement. In carrying this out it is always well to analyse the cause of the displacement in the particular fracture, as this will give a clue to the easiest method of reducing it, and will also advise us as to the probability of a recurrence of the deformity after reduction. In some cases the displacement is primarily due to the violence which caused the fracture, as, for example, in Colles's fracture. In such a case there is little if any tendency to the recurrence of deformity. A Colles's fracture if once successfully reduced does not tend to recur, and in most cases could be treated with perfect safety without any splint. In other cases the displacement is due to the pull of muscles, as, for example, in a fracture of the shaft of the femur. In such cases reduction has often to be carried out by means of extension, and it tends to recur if the extension is removed until such time as consolidation has taken place. In a third class the displacement is chiefly due to posture or to the weight of the limb, as, for example, in a fracture of the clavicle. In this case reduction is simple and the method of preventing recurrence is obvious.

The next point in the treatment of a fracture is the question of splinting. It is sometimes erroneously thought that those who advocate massage and movement in the treatment of fractures attempt to do without the use of splints. This is of course quite untrue. All that they ask is that splints should be used only for their proper purpose. This purpose is to prevent the recurrence of deformity after reduction. Splints should never be used to reduce deformity by pressure, and only occasionally should they be used to prevent deformity by posture. Their real use is to prevent such movements on the part of the patient as are likely to reproduce the displacement, and they are only necessary as long as such a recurrent displacement is possible. Just as recurrent displacement is more likely to occur in one fracture than in another, so splinting may be necessary in one case for a week, in another case for a month. In a Colles's fracture a splint is really scarcely ever necessary; it is often only used to prevent the patient using the hand; it can be removed for massage from the first, and can be discarded after a week or ten days.

In a Pott's fracture the splint can be removed for massage after a week, and can be discarded after about a fortnight. In a fracture of the femur the splint can generally be removed for massage and movement after fourteen to twenty-one days, but cannot be discarded before about the sixth week. The union of a bone by provisional callus is much more rapid than is often thought. Organisation of the effusion begins within the first forty-eight hours, and bony callus can be demonstrated in a skiagraph in from fourteen to twenty-one days. In one case of osteotomy in this hospital, treated by massage and without any splint, callus

was seen in a skiagraph taken upon the twelfth day.

To come now to the use of massage and movement. Massage can be carried on from the first, provided that no risk is run of disturbing the position. Similarly the only contra-indication for early movement of the neighbouring joints is a risk of thereby reproducing the displacement. This risk must of course vary not only with the individual fracture, but also with the manipulator, and also in the case of movements with the particular movements executed. Massage required for a fracture is a simple process and does not require any great amount of training; it can be readily carried out by the practitioner himself. He will thereby be able to arrange his splinting in such a way as to get at the part, and he will take the responsibility of any disturbance of position himself. The movements that are dangerous in any special fracture will be recognised if the cause of displacement in each case is analysed. For example, in a Colles's fracture there is practically no risk in carrying out any movement from the first. In a Pott's fracture it is well to avoid abduction and eversion of the foot in the early stages, as the structures on the inner side of the joint are torn and should not be stretched. In a fracture of the femur the weight of the limb must be carefully supported during the movements, and it is as well to keep up the extension to counteract the pull of the powerful thigh muscles. If each fracture is carefully considered in this way, and if the treatment is carried out by or under the supervision of the surgeon, practically all fractures can be safely treated by massage and movement from the first, with great benefit to the patient. Most of the opposition to these methods of treatment has arisen owing to misconception of the meaning of the terms "massage" and "movements" in relation to the treatment of fractures, and because it has often been the practice to leave this treatment in the hands of a professional masseur without any supervision.

April 9th, 1913.

DIPHTHERITIC HEMIPLEGIA.

By J. D. ROLLESTON, M.D.,
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HEMIPLEGIA is a rare occurrence in diphtheria. Out of 9075 completed cases admitted to the Grove Fever Hospital between August, 1899, and September, 1912, only six, of whom brief histories will be given, developed this complication. In all the initial attack was severe and was treated by large doses of antitoxin. All had abundant and persistent albuminuria. With the exception of Case 4, in which the hemiplegia was probably due to uræmia, all developed a well-marked cardiac lesion suggestive of an embolic origin for the hemiplegia, but in only one—Case 3—was it possible to verify this post mortem.

CASE 1.—Boy, æt. 6 years, admitted to hospital on November 21st, 1904, with very severe faucial and nasal diphtheria on the sixth day of disease. 24,000 units of antitoxin were given on admission and the same dose was repeated on the two following days. Albuminuria was present on admission and lasted till the forty-first day. November 25th: Voice nasal. Heart-sounds rapid and irregular. Liver enlarged. November 29th: Vomiting, which persisted till December 3rd. December 7th: Marked cardiac dilatation and irregularity. December 8th (twenty-third day of disease): Right hemiplegia and aphasia. December 10th: Ciliary palsy. December 29th to January 7th: Pharyngeal palsy necessitating nasal feeding.

Gradual improvement took place.

On discharge from hospital on March 23rd, 1905, he could walk and run, though with a marked limp. The fingers could be moved at will, but were unable to grasp any object. There were no wasting of muscles and no contractures. Speech was unaffected and there was no obvious mental deficiency.

CASE 2.—Girl, æt. 5 years, admitted to hospital on April 25th, 1905, with very severe faucial diphtheria on fourth day of disease. Twenty-four thousand units of antitoxin were given on admission and again on the following day. Voice nasal on tenth day. On fifteenth day the heart became dilated. On the seventeenth day right hemiplegia

and aphasia occurred. Death, preceded by coma, took place on the eighteenth day. No necropsy.

CASE 3.—Boy, æt. 3 years, admitted to hospital on April 15th, 1907, with severe faucial diphtheria on third day of disease. Eight thousand units of antitoxin were given on admission. On seventh day the heart-sounds were irregular, there was vomiting, and the liver was enlarged. On the fourteenth day right hemiplegia and aphasia occurred. Death on seventeenth day. Albuminuria was present on admission and lasted until death. Post mortem, in addition to extensive softening and liquefaction of the left cerebral cortex, two large infarcts were found in the left kidney.

CASE 4.—Girl, æt. 6 years, admitted to hospital on December 5th, 1910, with severe faucial diphtheria on third day of disease. Twenty thousand units of antitoxin were given on admission and again on the following day. Albuminuria was present from admission until death.

December 31st (twenty-ninth day of disease): Right hemiplegia of three hours' duration preceded by convulsions. January 2nd: Generalised convulsions. Blood-pressure 120 mm. Hg. January 4th: Death. No necropsy.

CASE 5.—Girl, æt. 3 years, admitted to hospital on May 20th, 1911, with very severe faucial diphtheria on third day of disease. Sixteen thousand units of antitoxin were given on admission and the same dose repeated on the following day. May 30th: Heart shows triple rhythm. June 3rd: Pain in right leg. June 4th: Left hemiplegia. Both legs cold; voice nasal. June 5th: Cyanosis of right lower extremity from foot to groin. Edge of liver two inches below ribs. Death the same day: no necropsy.

CASE 6.—Boy, æt. 5 years, admitted to hospital on September 3rd, 1912, with very severe faucial and nasal diphtheria. Twenty-four thousand units of antitoxin were given on admission and the same dose repeated on each of the two following days. Cardiac dilatation and irregularity occurred on the sixth day and was accompanied by vomiting, which persisted till the fifteenth day, and by enlargement of the liver. Knee- and ankle-jerks lost on the twenty-first day. Sudden onset of right hemiplegia and aphasia on the twenty-first day. Palatal palsy, shown by regurgitation of fluids through the nose, occurred on twenty-ninth day, ciliary palsy on thirty-third day, and pharyngeal palsy, necessitating rectal and nasal feeding, lasted from the thirty-sixth to the fifty-eighth day. Other palsies noted were paresis of rectal and vesical sphincters, and of the diaphragm during the occurrence of pharyngeal palsy. Albuminuria was present from the ninth to the forty-first day. No serum phenomena beyond slight urticaria of one day's duration on the nineteenth day.

On discharge from hospital on December 23rd, ninety-four days after the onset of hemiplegia, there were some spasticity of right arm and leg and slight right facial palsy, occasional slight tremor of the right hand, extensor plantor response, ankle clonus and combined movement of trunk and pelvis on right side. Intelligence was unaffected, but speech was limited to a few syllables. He could walk with very little support. When shown at the Clinical Section of the Royal Society of Medicine on January 10th, 1913, he was able to walk alone with a very slight limp, otherwise his condition remained unchanged.

My own experience of the rarity of post-diphtheritic hemiplegia is confirmed by a study of the literature. In 1905 I was able to collect only 65 cases, including the first two recorded above, and since that date 11 more have been published—Escherich (2 cases), Hecht, Moltchanoff, Jones and Hamilton (2 cases), Humphrey, Jeanneau, Burrows (2 cases), and Cubells—which, with my four cases that have occurred since then, make a total of 80.

Analysis of these 80 cases yields the following results: 28 were males, 37 females, and in 15 the sex was not stated. The ages ranged from one and a half to seventeen years. Right hemiplegia occurred in 48, in 21 of whom aphasia was noted, left in 27, and in 5 no details were given. The occurrence of the hemiplegia took place at the following dates: In the first week 1 case, in the second week 14, in the third 27, and in the fourth to the seventh 12. In 14 cases, where no exact date is given, it is said to have developed in convalescence; in 12 no date whatever is given. Recovery took place in 52, death in 24, in 4 no details were given.

Necropsies were held in 18 cases. Embolism was found in 13, thrombosis in 3, hæmorrhage in 1, and sclerotic atrophy in 1.

In all the cases where details are given the initial attack was severe.

Albuminuria and ordinary diphtheritic paralyses were present in a large number.

Diphtheritic hemiplegia is usually due to embolism, evidence of which has been found in twelve out of eighteen autopsies. In a large number of the recorded cases who did not come to autopsy or who recovered it is noted that there was severe cardiac involvement.

Embolism is probably commoner in diphtheria than in any other acute contagious disease, being due, as Marfan has pointed out, to an apical endocarditis. The emboli may be found not only in the cerebral circulation, but in the kidneys, spleen, lungs, and even the abdominal aorta, as in Marfan's case. In my recent case of gangrene of the leg following diphtheria the condition was due to femoral embolism, and was probably associated with emboli in the spleen, kidney and opposite leg though the diagnosis of multiple emboli could not be verified, as the child recovered.

Case 5 (*vide supra*) was also probably an example of multiple embolism, though here again this diagnosis was not confirmed as a necropsy could not be obtained.

The prognosis in diphtheritic hemiplegia as in hemiplegia generally following acute infections is unfavourable as regards complete recovery. There are only four cases on record in which complete restoration of function has occurred (Levi). In the great majority of cases contractures and atrophy have supervened in the paralysed limbs.

Hemichorea and imbecility are other sequelæ occasionally met with.

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April 9th, 1913

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from page* 352.)

(170)

Dangers of the Arylarsonates.

Atoxyl and other arylarsonates have been vaunted as ideal preparations, but I have witnessed two cases of temporary blindness and one of persistent headache following their use; they are, therefore, not to be recommended to any practitioner who has to live upon his reputation.

(*Ibid.*, Mr. McDonagh, 'Clin. Journ.,' vol. xxxvi, p. 190.)

(171)

The Inter-menstrual Period,

or

Physiologically, Nature works slowly but surely.

The inter-menstrual weeks are not in any true sense weeks of repose from the preparation for reproduction. They are merely weeks of quiet and unobtrusive marshalling of forces which concentrate for the next molimen, and although the marshalling is unaccompanied by any objective sign, its work proceeds as regularly and inexorably as the tides on the sea-shore. Menstruation is too often regarded as if it were a spasmodic explosion, but Nature, in her physiological moods, dislikes a hurricane as cordially as she abhors a vacuum.

("The Cardiopathies of the Menopause," by Leonard Williams, M.D., M.R.C.P., 'Clin. Journ.,' vol. xxxiii, p. 325.)

(172)

Rectal Symptoms demand a Rectal Examination.

The mistake which is made is this: Men very often forget, or omit, to examine a patient *per rectum* when examining him for *rectal symptoms*. If you are not going to pass cases of carcinoma by, you should make it a rule whenever patients come under observation and they complain of piles or the passing of blood *per anum*, or with the stools, never to omit to make a complete digital examination of as much of the rectum as can be investigated with the finger. Also in these cases where there is nothing abnormal to be made out in the

form of piles, and the digital examination reveals nothing pathological, and there is still evidence of the patient passing blood *per anum*, or having occasional attacks of diarrhœa, followed by the passage of blood or slime, do not be content with merely investigating by the finger from below, but have the patient anæsthetised, and have the upper portion of the rectum and lower portion of the pelvic colon examined by the sigmoidoscope. If you do this you will not miss a case of carcinoma in the upper part of the rectum, but if you do not examine these cases in that way you are very likely to miss them.

("Two Cases of Carcinoma of the Rectum treated by the Abdomino-perinæal method," by H. J. Waring, M.S., F.R.C.S., 'Clin. Journ.,' vol. xxxvi, p. 193.)

(173)

The Value of Doubt.

A philosophical historian has said that we should learn first of all to doubt, because doubt leads to inquiry, and inquiry leads to discovery. And if you feel doubt about every case which is presented to you I am sure you will not fall into some of the pit-falls that others have fallen into.

("Medical Cases in Surgical Practice," by C. B. Lockwood, F.R.C.S., 'Clin. Journ.,' vol. xxxiii, p. 49.)

(174)

In Difficult Cases reserve your Diagnosis.

I often wonder why anyone should suppose himself able to arrive at a definite and positive conclusion about complicated conditions after a short examination in the consulting room. If you are in doubt about a case, say that you would like to see it again and have time to think it over, and have an opportunity of examining it on a future occasion.

("Femoral Ruptures and their Management," by C. B. Lockwood, F.R.C.S., 'Clin. Journ.,' vol. xxxv, p. 209.)

(175)

Femoral Ruptures,

or

Text-book Teaching v. *Clinical Diagnosis.*

In the diagnosis of femoral ruptures you may be misled by considerations of sex. For instance, you are taught that inguinal ruptures are common in men and femoral in women. But that is a consideration which need not weigh in your minds, because the question is not what is common in men, or what is common in women, but what is the matter with the patient who is in your presence.

(*Ibid.*)

(176)

To the Careless any mistake is Possible.

I operated upon a woman who was wearing a femoral truss on a dilatation of the saphenous vein. You would think that that was an impossible mistake for anybody to commit. But anything is possible to those who do not properly examine their patients.

(*Ibid.*)

(177)

"Freak Trusses."

Again I advise you to have nothing to do with freak trusses. The best type of truss that you can select and learn to understand is the ordinary spring truss.

(*Ibid.*)

(178)

One Cause of Nerve Atrophy.

Prolonged forced functioning of any nerve structure tends to result in atrophy of that structure.

("Tabes Dorsalis, Pachymeningitis Spinalis, Cerebellar Tumour and other Disorders of Gait," by Thomas D. Savill, M.D.Lond., 'Clin Journ.,' vol. xii, p. 21.)

(179)

In Anomalous Skin Eruptions think of Drugs.

The inability to classify the cutaneous phenomena as belonging to any recognised skin affection should put the practitioner on his guard, and is frequently sufficient ground for the dermatologist to diagnose a drug eruption. The polymorphism of such an eruption may, therefore, be of great help in making a diagnosis.

("Skin Rashes and their Treatment," by J. L. Bunch, M.D., D.Sc., M.R.C.P., 'Clin. Journ.,' vol. xxxvi, p. 257.)

(180)

Antiquity of Arthritis Deformans.

We have not very definite records as to the date at which this disease of the synovial membranes and cartilages first appeared, but we know that arthritis deformans is not a disease of mushroom growth such as appendicitis and appendicitic dyspepsia, for some bones of men who lived about 3700 B.C. demonstrate the characteristic changes of this disease.

("Arthritis Deformans," by Otto Grunbaum, M.D.Cantab., F.R.C.P.Lond., 'Clin. Journ.,' vol. xxxvi, p. 290.)

(181)

In the Multitude of Remedies there is Weakness.

The treatment of arthritis (deformans) is so manifold that we may safely conclude none is of much avail.

(*Ibid.*)

(182)

Anti-Vaccination,
or
Government by a Minority.

It is a common criticism on the combined civilisation and sentimentality which rule this country that the prejudices of a minority of people are still able to expose the majority to occasional outbreaks of a very repulsive filthy disease, which we have now known how to prevent for a hundred years. If we could all make up our minds to be logical and practical, and insist on the regular performance of vaccination and re-vaccination, we should be able to laugh at smallpox, and save the money of the rate-payers by abolishing the isolation hospitals which to-day exist for smallpox cases.

("Smallpox and its Early History," by F. M. Sandwith, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvi, p. 294.)

(183)

An Object Lesson for the Conscientious Objector.

I believe that any conscientious objector could at once be converted to belief in vaccination if he could see, as I have done, healthy children living for weeks in a smallpox hospital, and obliged to be there because there was no one else to look after them. One thing, and one thing only protected these children—they were vaccinated directly their mothers were found to have smallpox.

("The Value of Vaccination," by F. M. Sandwith, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvi, p. 346.)

(184)

Limitations of Mercury and Iodide of Potassium in Syphilis.

If a gumma destroys nerve-cells or nerve-fibes no amount of mercury or iodide of potassium will replace the structures which have disappeared.

("A Clinical Demonstration at the Polyclinic," by Purves Stewart, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvi, p. 353.)

(185)

Many of us Live to Eat rather than Eat to Live.

As for the public, the pleasures of the table, and habitual strayings from the paths of physiological righteousness, are, as we know but too well, powerful rivals of such prudential considerations. Modern cooking, with its alluring refinements, tends to make *gourmets*, and, what is infinitely worse, *gourmands* of us all. Colour and fantastic form in dress are well matched by the varied and tempting flavours, imparted nowadays, to cooked food. The mischief is further aggravated by the fact that the hurry-scurry of twentieth century life renders us precipitate and preoccupied feeders.

("Note from a Foreign Spa," by John H. Barnard, M.D. (of Contrexéville), 'Clin. Journ., vol. xxxvii, p. 14.)

(186)

The Typical Buveur.

Viewed in profile, the typical *buveur* is a sad sight, both æsthetically and physiologically. Hyperalimentation and physical indolence—a want of balance between income and expenditure—are reflected in the rotundity of his outlines. Enteroptosis and unwieldiness are not infrequently his more striking characteristics. These, with the easily aroused dyspnœa of hypertension, make up a picture of preventable, premature senility that is discouraging to the conscientious practitioner, and which, moreover, does not increase one's respect for human intelligence. See him at table, and the gusto with which he "tucks in" affords an easy explanation of his early physical decadence.

(*Ibid.*)

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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SOME CERVICAL CYSTS OF CONGENITAL ORIGIN.*

By G. H. EDINGTON, M.D., F.R.F.P.S.G.,

Assistant Surgeon, Western Infirmary; and Extra Honorary Surgeon, Royal Hospital for Sick Children, Glasgow.

As the cases which I am about to describe are examples of congenital abnormality in the neck, it will be necessary briefly to refer to the anatomy and development of this region. If the embryo at the end of the third week be examined, it will be found that in the lateral wall of the foregut there are four depressions running dorso-ventrally. These depressions, termed "pharyngeal pouches,"† extend outwards, pressing aside the mesoderm, to come in contact with the ectoderm. When the pharyngeal endoderm reaches the ectoderm the two fuse to form the "epithelial closing membrane." At the time that the membrane is formed, and *not before*, external depressions make their appearance. They are known as the "branchial grooves." A later ingrowth of mesoderm takes place and separates the grooves from the pharyngeal pouches.

While in some animals, *e.g.* fishes, the epithelial closing membrane disappears, so that an actual cleft is formed, in man the occurrence of such a cleft is not usual, so that in the human subject there is not at any time in the normal course of development a communication between the cavity of the pharynx and the surface of the neck.

The areas between the successive grooves and pouches, retaining as they do the mesoderm, form a series of arches or bars which are known as "branchial" or "visceral arches." Of these the first two have received special names. The first is

* A communication made to the Glasgow Eastern Medical Society, March 5th, 1913.

† Keibel and Mall, 'Manual of Human Embryology,' vol. ii, pp. 446 *et seq.*

called the "mandibular," and the second the "hyoid" arch, and from them respectively are formed the mandible and the styloid process, stylo-hyoid ligament, and lesser cornu of the hyoid bone. The remaining arches, three in number, are collectively named "branchial" arches. From the first of them comes the greater cornu, and also, after fusion with the ventral part of the hyoid arch, the body of the hyoid bone.

As growth proceeds the mandibular and hyoid arches outstrip the branchial arches, which lag behind. The former two come to overlap the latter, and a recess is formed—the "sinus præcervicalis" of His. The external opening of the sinus closes, and the shut-in cavity is connected by diverticula (the former branchial grooves) with the second and fourth pharyngeal pouches.* The buried sac and diverticula ultimately collapse and their epithelium disappears. The first branchial groove, however, persists in part, and its dorsal portion forms the external auditory meatus. Traces of the pharyngeal pouches remain in the fully developed pharynx. Thus, the Eustachian tube, terminating in the tympanic cavity, represents the first pouch, and the supra-tonsillar pouch the second.

Development of median thyroid.—The median thyroid is developed as a bud from the endoderm at the ventral ends of the second pharyngeal pouches.† The bud arises between the buccal and pharyngeal portions of the tongue during the third week, and grows downwards and backwards, bifurcating to form a network of acini—the isthmus and part of the lateral lobes of the thyroid gland. Its point of origin is represented by the foramen cæcum of the tongue. The remainder, the thyro-glossal tract or duct, disappears at the commencement of the second month. Traces of its lower end are seen sometimes as a "pyramid," passing from the isthmus towards the hyoid bone. The body of the hyoid bone is developed in the track of the duct, and splits the latter.

Formation of epithelial "rests."—From the above it will be seen that in the course of development of the neck there are three structures, or sets of structures, composed partly of epithelium, which are buried in the deep tissues, viz. branchial grooves, sinus præcervicalis and thyro-glossal duct. Normally these structures are obliterated, but as may readily be understood, epithelial "rests" may arise from incomplete obliteration. From such "rests" cystic formations may originate, and the cases which I shall now relate are to be looked upon as having arisen in this way.

Case 1: *Cyst of sinus præcervicalis.*—This case was that of a married woman, æt. 32 years, on whom I operated in April of last year.

The patient was admitted to the infirmary on account of a prominent ovoid swelling in the supra-sternal region, and overlapping the manubrium. The swelling was first noticed six years previously and slowly increased in size, without causing any pain. It was incised by her doctor, and whiteish, porridge-like matter evacuated. After the wound had healed the swelling re-formed, and for a year previous to my seeing her it was slightly painful. The situation and appearance of the swelling are shown in the photograph (Fig. 1), taken immediately before operation. The swelling was fluctuant, and no processes could be felt passing off from it.

A transverse incision exposed a cyst under the

Fig. 1.—Case 1. Cyst of sinus præcervicalis.

* Keibel and Mall, *loc. cit.*, p. 456.

† Quain's 'Elements of Anatomy,' eleventh edition, vol. i, p. 168, 1908.

deep fascia in Burns's space.* The lower half of the cyst rested on the front of the manubrium ; the upper passed deeply backwards between the sterno-mastoid muscles to lie on the sterno-hyoid muscles. Behind, it had a close relationship with the anterior jugular vein of the left side. The cyst, which was removed entire, measured 6 cm. vertically by 4·5 cm. transversely.

Microscopical characters.—The specimen was examined by Dr. Haswell Wilson, who found that the contents were sebaceous material and cholesterin crystals. The wall of the cyst was composed of an outer fibrous capsule, lined by stratified squamous epithelium, underlying which there was a cellular tissue containing giant-cells.

Remarks.—As regards the diagnosis before operation, the physical characters of the swelling did not distinguish it from a softened tuberculous gland, such as is occasionally met with in this situation. There was an absence, however, of enlarged glands elsewhere in the neck, or of any scars which would point to the lesion being of a tuberculous nature. The history, also, was suggestive of a sebaceous cyst. That it had no connection with the thyro-glossal duct was evident from the absence of a cord extending up to the hyoid bone (see below).

Its situation under the deep fascia which separated it from the skin of the part suggested its origin from an embryonic or fœtal inclusion. In view of what is known regarding the development of this region, the cyst may most properly be considered as having its origin in a "rest" of the sinus præcervicalis.

CASE 2 : *Lateral cyst of branchial origin.*—The patient in this instance was an unmarried woman, æt. 28 years, who was the subject of a large swelling in the left side of the neck. The swelling had been present for a year or two, and on two occasions it had burst, and after discharging for a time had healed.

The swelling (Fig. 2) was of peculiar shape, conical, and bulged downwards, forwards and outwards from the region of the angle of the mandible. At the apex of the swelling the skin was much thinned, and pus was visibly shining through the cuticle.

An incision in the thinned area opened a superficial abscess which communicated with the cavity of a sac having a distinct wall. A finger introduced into the cavity of the sac passed upwards and struck the angle of the mandible, the mastoid process, and the deep vessels of the neck—an extensive relationship of the sac-wall suggestive of that of a hydrocele.

FIG. 2.—Case 2. Branchiogenic cyst.

Portions of the sac-wall were removed for microscopical examination, and the cavity was packed, and afterwards allowed to contract down over a drainage-tube. Dr. Browning reported that the sac-wall consisted of connective tissue, with, in places, lymphoid tissue, and a lining of stratified squamous epithelium. The pus was sterile.

One month after the first operation the fistulous track was exposed by an incision along the anterior edge of the sterno-mastoid muscle. It was found impossible on account of adhesions to dissect out with safety the tube of tissue, until it had been slit open. After being slit the wall was partly dissected and partly peeled off the jugular vein. The upper end seemed at first to be in relationship with the tonsillar region of the fauces, but as the dissection proceeded it was found that the cord passed backwards more to the transverse process of the atlas, to which region it was adherent by a fibrous band. The upper end of the cord was covered by the posterior belly of the digastric muscle, but there were also adhesions to it from the outer surface of

* Allan Burns, 'Observations on the Surgical Anatomy of the Head and Neck,' 1811, p. 5.

this muscle. The dissection was difficult on account of the infiltrated state of the tissues surrounding the wall of the tract. The inner surface of the wall generally was smooth but showed many buds of granulations.

The tissues removed were examined by Dr. Shaw Dunn, and showed the characters of a cyst-wall consisting of fibrous tissue, lined by stratified squamous epithelium which lay closely over a narrow layer of lymphoid tissue. Yellowish material in the wall proved to be chronic inflammatory tissue, containing abundant "foam" cells loaded with fat.

Remarks.—The size and shape of the swelling clinically were unlike what is usually met with in the case of tuberculous gland enlargement. If the condition had been tuberculous the likelihood is that there would have been multiple points of softening over the large mass. That the condition was a cyst was at once seen when the wall had been exposed. Further, the rambling nature of the cavity suggested its congenital origin.

The operation for removal was done in two stages so as to avoid opening up the neck in presence of suppuration and infected cyst-contents, and it was felt also that by allowing the parts to collapse extirpation could be effected through a smaller incision.

The relation of the upper end of the tract to the digastric muscle was characteristic of a branchiogenic cyst arising in connection with the second groove; but its attachment to the atlas was unusual, and is perhaps to be accounted for by the adhesions associated with the precedent suppuration.

It is not possible absolutely to say that the sinus præcervicalis had no part in the formation of the "rest" from which the cyst sprang, but if we may judge from the high situation and attachments of the sac it seems more probable that it originated from the branchial groove.

Case 3: *Thyro-glossal duct fistula.*—Elsie J—, æt. 6 years, was sent to me by Dr. Moorhouse, of Stirling, in December, 1911, on account of a discharging sinus in the neck a little above the manubrium.

Two years previously a raised lump made its appearance at this spot. One year later a "boil" seemed to form and burst, discharging greenish matter. Since then the swelling had gathered and burst about once a fortnight.

Fig. 3.—Case 3. Thyro-glossal duct fistula.

Examination showed in the median line at the root of the neck a depressed pit of scar-like tissue 7 mm. in diameter, overhung by a transverse fold of skin measuring 1·3 cm. from side to side (Fig. 3). When the child swallowed the depression was pulled up under this fold. On palpation a cord extending up to the hyoid region was felt slightly to the left of the median line.

Operation.—On December 21st, 1911, a transverse incision was made exposing the body of the hyoid bone and the infra-hyoid region, but the upper end of the cord was not identified. A transverse elliptical incision was made round the fistulous opening. The upper lip of this incision was retracted upwards, and the cord leading from the fistula freed from its surroundings. The space between the two incisions was then tunnelled by a dressing-forceps. The fistula and tract were pushed up through the tunnel, and brought out through the upper incision. The upper end of the tract was found to pass behind the body of the hyoid bone, to which it was firmly attached. A piece of the body of the bone was separated by bone-pliers, and removed along with the attached tract. A fascial band extended from the lower end of the fistulous area towards the manubrium, and was divided during removal. The tract tapered from below upwards (Fig. 4) from 7 mm. across at its

lower end to 3·5 mm. where it joined the hyoid bone. It measured 3·5 cm. in length.

Microscopical characters. — Transverse sections of the tract a little below its upper end showed it to be a canal, with a wall of dense fibrous tissue. The lining, which presented longitudinal folds, consisted of stratified epithelium, the inner layer of which was columnar ciliated. The lining was separated from the fibrous wall by loose cellular tissue.

The wounds remained healed till the middle of February, 1912. At that time a little "lump" appeared in the middle of the upper scar, broke down, exuded some secretion for a week, and then healed. One week later this sequence of events was repeated. The scar remained dry from the middle of March till the middle of April, when the small lump again made its appearance, broke down, and discharged as before.

The patient was photographed on May 23rd, 1912 (Fig. 5). In the centre of the upper scar of the first operation there was a small rounded projection, covered by reddened skin, and measuring ·5 cm. in diameter. On swallowing, this was

Fig. 4.—Case 3. Fistulous tract excised. At lower end is fistulous opening in puckered skin; at upper is portion of hyoid body.

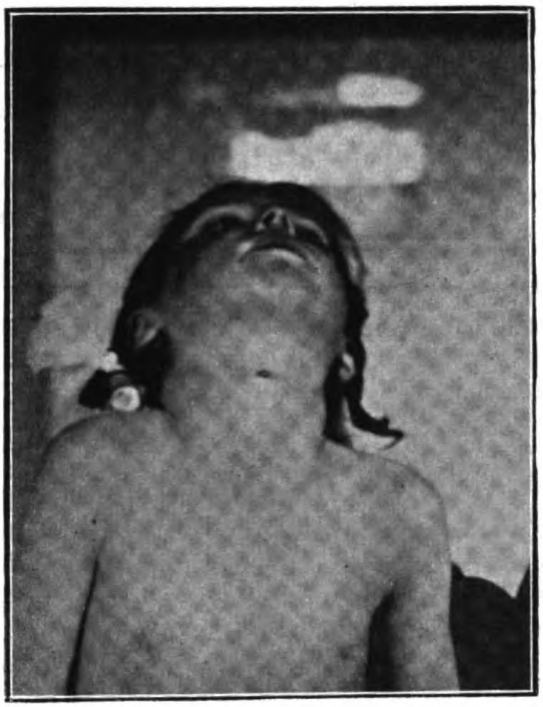

Fig. 5.—Case 3. Recurrent fistula at level of hyoid body.

pulled backwards and upwards, and clearly was fixed to the remains of the body of the os hyoides. The lower scar was almost invisible. On this date the mass was surrounded by an elliptical incision and dissected off the thyro-hyoid membrane. The portion of hyoid body remaining from first operation was also removed, leaving a fibrous bridge (? periosteum) for the attachments of the genio-hyoid and genio-hyoglossi muscles. The sterno-hyoid muscles were approximated across the middle line by a single catgut suture, and the skin edges were closely sutured, without drainage.

Microscopical characters. — Vertical antero-posterior sections of the spherical lump showed a cavity lined by granulation-tissue. No traces of epithelium were to be found.

After-history.—Dr. Moorhouse wrote me on June 2nd that the wound was soundly healed; and again on February 18th, that it had remained healed and given no further trouble. The upper scar is drawn upwards on swallowing.

Remarks.—The diagnosis in this case presented no difficulty, the cord extending up to the hyoid bone, being practically proof positive of the existence of a persistent thyro-glossal duct. The pulling up of the fistulous opening during swallow-

ing demonstrated its relationship to the cord, and at the same time established its true nature.

The course of events after the first operation illustrates the difficulty of complete removal of the remains of the unobliterated thyro-glossal duct in the hyoid region; and the cure which resulted from the removal of the remains of the body of the hyoid bone is an indication of the radical measures which are necessary. "Rests" of the median thyroid do not always take the form of an elongated cord. Sometimes they are quite short and confined to the hyoid or thyro-hyoid region. This is exemplified in the next case.

CASE 4: *Cyst of thyro-hyoid region.*—A man, æt. 44 years, was sent to see me by Dr. Hugh McLean on April 1st, 1910. He had a swelling over the larynx, noticed about one month previously and not giving rise to any trouble.

A rounded sessile swelling of considerable hardness was present over the upper part of the larynx. It was exposed by a transverse incision, and found to be a cyst lying in the thyro-hyoid space under cover of the sterno-hyoid muscles: these were partly cut through before the cyst was reached. The upper part of the cyst was adherent to the posterior surface of the body of the hyoid bone; from its lower part large veins passed downward in the neck. It was dissected out entire.

After hardening in formalin the cyst measured 1·25 cm. in diameter. On incising it brown gelatinous matter escaped. Under the microscope this material was found to contain cells (? epithelial) and leucocytes, all more or less fatty.

Sections of the wall showed it to be composed of fibrous tissue with a lining of stratified epithelium. The lining cells had been much flattened from intra-cystic pressure, and were apparently of squamous or subcubical type.

Remarks.—The great hardness of the cyst was suggestive of the swelling being solid; and this, added to its immobility, led me to fear that I had to deal with a malignant growth. It was only during the operation that the anatomical relationship to the body of the hyoid bone gave a clue to the true nature of the swelling, and this was confirmed by microscopical examination.

Such cysts are looked on by Keith * as arising from "remnants of the duct or of secondary detached acini of the thyroid," and though more commonly found between the genio-glossi muscles, "they may also occur between the hyoid and thyro-hyoid membrane"; but he adds that sometimes bursal enlargements are mistaken for cysts of thyroid origin. In the present case the distinction in favour of the latter is drawn from the epithelial lining.

While they may properly be looked upon as connected with the development of the median thyroid, they differ *clinically* from those cysts associated with a cord-like persistent thryo-glossal duct, in that they are much more easily extirpated.*

Dr. McLean informs me that he saw the patient a few days ago, and that there has not been the slightest appearance of recurrence.

April 14th, 1913.

* Keith, 'Human Embryology and Morphology,' third edition, p. 245, 1913.

Anti-diphtheritic Serum in Hæmophilia.—M. J. Perkins ('Journ. Amer. Med. Assoc.')—A boy, æt. 16 years, was admitted to hospital on Thursday, July 18th, 1912, at noon. He had been bleeding for ninety hours from a cut just above the right upper central incisor, due to striking a bracelet on a young woman's arm when falling out of a hammock. Two of his mother's brothers suffered from hæmophilia. He was bleeding profusely, and much weakened from loss of blood. He was given 3000 units of anti-diphtheritic serum hypodermically, no other horse-serum being at hand. A piece of absorbent cotton was placed over the cut, and the lip drawn down over this. There was a little bleeding after this, until 7 p.m., when pressure was applied, and 30 gr. of calcium lactate were given by mouth. There was slight bleeding between the teeth until the following morning. On Friday and Saturday he was given 180 gr. of calcium lactate. The bleeding was easily controlled by placing a piece of absorbent cotton between the gum and lip; hæmorrhage was less each time the cotton was changed, and on Saturday evening it had ceased.

* Edington, 'Brit. Med. Journ.,' October 1st, 1904.

SIX CLINICAL LECTURES*

ON

THE DIAGNOSTIC VALUE OF SENSORY CHANGES IN DISEASES OF THE NERVOUS SYSTEM.

By HENRY HEAD, M.D., F.R.C.P., F.R.S.,
Physician to the Hospital.

LECTURE VI.

IN this my last lecture, I shall follow the afferent impulses from the optic thalamus to the cerebral cortex, and conclude with a description of the changes which may occur when sensation is disturbed from purely psychical causes.

We have already seen in the previous lecture that sensory impulses pass in two directions from the receptive junction in the optic thalamus—firstly, to the grey matter of the thalamus itself, and secondly, by way of the internal capsule to the cortex. (Fig. 16, Lecture V.†) Every afferent impulse must pass through this narrow neck before it can affect the cortex directly, and at this point the sensory paths are gathered so closely together that a lesion in this situation produces gross sensory loss on the hemiplegic half of the body.

The form assumed by this disturbance of sensibility depends on the position of the lesion; if it is low down, on a level with the receptive junction in the optic thalamus, both structures will almost certainly be affected together. The loss of sensation consequent on such combined lesions of the thalamus and internal capsule is particularly gross, consisting of more or less complete loss of appreciation of touch, pain, heat, cold, posture, weight, size and shape; in fact, the patient will be gravely hemiplegic, with severe loss of sensibility over the whole paralysed half of the body. The similarity in blood-supply and the close juxtaposition of the thalamus and capsule make such lesions particularly common, especially if the disease is of vascular origin.

But, whenever the lesion affects the sensory paths of the internal capsule after they have left the thalamus, the changes in sensibility are not only less severe, but are demonstrably different in quality; some of them are easily determined, while others will puzzle you considerably unless you are aware of their nature.

Let me, then, show you a patient in whom the lesion is believed to lie in the internal capsule after the paths to the essential grey matter of the optic thalamus have been given off.

CASE 10.—Here is a man, æt. 35 years, who has long suffered from mitral stenosis of rheumatic origin. Some months ago he was suddenly struck with left hemiplegia, evidently produced by an embolus. Now, the vessels which supply the internal capsule are the commonest site for cerebral embolisms, and there can be little doubt that in this case, the lesion lies in the internal capsule. At the present time he shows a flaccid hemiplegia; both right arm and leg are affected, and all movements of the fingers are impossible, but there is some power at the elbow and slightly more at the shoulder-joint. He shows the usual exaggeration of the reflexes in the right arm and leg, and the plantar reflex on this side gives an extensor response.

The severity and extent of the paralysis with so small a lesion as an embolus evidently point to the internal capsule as the site of the lesion, and an examination of the sensory changes bears out this diagnosis.

He can appreciate all the coarser sensory stimuli, and a prick or the more intense degrees of heat and cold, cause the same sensation on the two halves of the body; but there is no over-response such as we saw so strikingly in cases of thalamic lesions.

Nevertheless, you will notice that when he attempts to find the finger of the affected hand with the normal one he is grossly at fault—in fact, the left arm may be moved from one position to another when his eyes are closed, and he is completely ignorant that its position has been changed. This can be demonstrated in a striking manner by the following procedure. After he has closed his eyes, I move the affected arm or leg into some fresh position, asking him to imitate the posture it has assumed. Usually he makes some rough attempt at imitation with the sound limb, but directly he is allowed to open his eyes the grossness of his error causes a smile of surprise to pass over his face—striking evidence of the extent of his want of postural recognition. He has also lost all power of appreciating size, shape,

* Delivered at the London Hospital.

† See CLINICAL JOURNAL, October 2nd, 1912, p. 409.

form or weight in the affected hand, but the vibrations of the tuning-fork evoke a sensation, though less distinctly than on the normal limb.

So far the sensory changes are not hard to elucidate, but any attempt to examine tactile sensibility meets with considerable difficulties. Pressure with the finger or other hard object is certainly appreciated, but unless the cotton-wool is rubbed firmly and repeatedly over affected parts of the body, he not infrequently fails to recognise the stimulus altogether. You will have noticed that, although he responded to all the contacts with cotton-wool over the normal hand, similar stimulation of the affected hand evoked at most three answers. Directly we use instruments of precision with which the strength of the tactile stimulus can be measured, it is found that it must be stronger on the abnormal side in order to produce a complete series of responses—that is to say, tactile sensibility is not destroyed, but the threshold is materially raised. In the same way, we can show that it requires a somewhat higher temperature on the abnormal side to produce the sensation of heat, and a somewhat lower temperature before he can appreciate with certainty that the stimulus is cold.

Thus, we can say that at this level of the nervous system the main disturbance of sensibility consists of loss of recognition of posture, passive movement, size, shape and weight, together with a distinct raising of the threshold for tactile and thermal stimuli. The pain threshold, however, is entirely unaffected.

Thus, of all the afferent impulses that entered the central nervous system, one group after another has diverged to its receptive centres, and finally, after the internal capsule has been traversed, those only remain which will subserve the cortical aspects of sensation. Let us consider for a moment the grouping of these impulses, impulses which form the materials with which the grey matter of the cortex has to deal. The most important group consists of those concerned with the recognition of posture and passive movement; a second group underlies certain aspects of tactile sensibility, whilst on a third group depends the power of appreciating two points applied simultaneously to the skin and the recognition of size and shape; a fourth group underlies localisation of the stimulated spot, and a fifth group is concerned with thermal sensibility. This grouping of afferent impulses on their way to the cortex has been determined by many observations on cases where the lesion lay at this level of the nervous system.*

Out of these five groups of afferent impulses the activity of the *cortex* manufactures the physiological equivalent of certain aspects of sensation; what aspects of sensation are the consequence of cortical activity can be discovered by studying a case such as that of the man I shall now show you in whom a portion of the cortex was removed many years ago.

Case 2.—He is æt. 48 years, and on October 31st, 1905, a large gumma was removed from the inferior parietal lobe on the right side.† He is now in active work and perfect bodily health except that his left arm is weak and he drags his left leg slightly. The reflexes on the left side are somewhat exaggerated, but the plantar gives a flexor response and no ankle clonus can be obtained. Thus, as far as motor and reflex action are concerned he is a case of hemiparesis, with some hypertonicity but no gross affection of the pyramidal tracts.

Let us now examine the condition of sensation on the affected half of the body. He tells us that when he wakes at night he frequently does not know he has a left arm until he finds it with the normal hand. That is to say, the psychical preformations and representations, by which we know we have an arm, are so little present that, if he wakes in the dark, he is obliged to reconstruct them by touch. In this way, the sensory defects due to a cortical lesion may differ from those produced by disturbance of the internal capsule and afferent paths; such a lesion cuts off the incoming impulses only whilst a destruction of the cortical centre removes the station to which these impulses are going. In the one case, the patient remains conscious of the existence of his limbs, though he does not know where they lie, whilst with lesions of the cortex he may lose all consciousness of the existence of the affected parts.

This loss of recognition of the posture of his affected hand leads not only to erroneous pointing

* *Vide* Head and Holmes, 'Brain,' vol. xxxiv, p. 174.

† This case is given in full in the paper by Head and Holmes, *op. cit.*, p. 240. He is now under the care of Dr. Gordon Holmes, to whose kindness I am indebted for his presence here to-day.

with the normal forefinger but to the following characteristic difficulty. If, whilst groping for the index of the affected hand, he happens to find some other part of it, he cannot tell on which side the finger lies which he is seeking, and is as likely as not to grope towards the wrong part of the hand. From sensations in the normal hand he knows he has grasped the other hand, but he is ignorant of how to pass from the point of contact to the finger he is seeking.

When, however, he is asked to point with the affected hand to the normal index he is able to do so without difficulty, because he knows the position of the point at which he is aiming. Moreover, the abnormal hand, although paretic, is an unexpectedly effective indicating instrument, because both loss of power and loss of sensation are always less at the shoulder than at the distal parts of the limb in cases of cortical defect. Thus, the good hand seeking the bad one does not know where it is to be found, and so makes a bad record; on the contrary, the affected hand makes a better record when seeking the normal forefinger, because the position of the goal is known. You can make a tolerable score with a bad rifle if you know the position of the target; but a perfect rifle will not enable you to score if you do not know where to shoot. This is an important point in differential diagnosis, as we shall see when we consider psychical changes in sensibility.

The next change in sensation is revealed when the affected hand is repeatedly stimulated with cotton-wool. At first the patient did not respond to the touch, but all of a sudden he began to answer correctly; then he ceased to respond to the same stimulus, but after a series of failures again began to respond perfectly. This mode of answering to an approximately constant stimulus occurs normally when its strength lies close to the threshold. But in cortical disease this uncertainty of response is present even with tactile stimuli of several grammes. The answers are irregular and uncertain, and increasing the stimulus does not of necessity lead to an equivalent improvement in the proportion of accurate replies. There is no absolute loss of tactile sensibility to stimuli below a certain strength, as in cases where afferent paths are destroyed at the internal capsule or elsewhere, but the answers are irregular and uncertain, fatigue is induced with unusual facility—in fact, when the affected parts are being tested, the patient seems to be untrustworthy.

He is unable to appreciate the form or relative size and weight of objects placed in his affected hand, and the power of localising the spot touched is also abolished. Let me show you a method of recording this failure of localisation which avoids the difficulty of pointing to a hand of whose position he is ignorant. The hand to be tested—the left—is hidden from the patient's sight with a screen. My assistant, standing behind him, places his own left hand in a similar position in full view of the patient, who is asked to indicate on this living model the spot where he believes he has been touched by me. I then mark on a life-sized diagram both the point I have touched and that to which the stimulus was referred by the patient. This method does away with all the technical difficulties of spot-naming and the fallacies of the groping method. By this means it is obvious that, although our patient can localise the position of the spot touched with perfect accuracy on the normal side, he is grossly at fault on the affected hand; in fact, he frequently gives up all attempts saying, "I do not know."

I cannot leave these sensory abnormalities produced by a cortical lesion without saying a few words as to their meaning. We believe that the cerebral cortex is the organ by which we are able to focus attention upon the changes evoked by sensory impulses. A pure cortical lesion, which is not advancing or causing periodic discharges, will change the sensibility of the affected parts in such a way that the patient's answers appear to be untrustworthy. Uncertainty of response destroys all power of comparing one set of impressions with another and so prevents discrimination.

But, in addition to its function as an organ of local attention, the sensory cortex is also a storehouse of past impressions. These may rise into consciousness as images, but more often, as in the case of special impressions, remain outside central consciousness. Here they form organised models of ourselves which may be termed "schemata." Such schemata modify the impressions produced by incoming sensory impulses in such a way that the final sensations of position or of locality rise into consciousness charged with a relation to something that has happened before. Destruction of

such schemata by a lesion of the cortex renders impossible all certain recognition of posture or of the locality of a stimulated spot within the affected parts of the body.

Let us now pass to the *psychical level* and examine the nature of the changes in sensation produced from purely mental causes. Babinski holds that all hysterical manifestations are artefacts, and that most of them are feigned or suggested by medical examination. I hope to show you that whatever sensory changes occur in hysteria follow a mental and not a physiological or anatomical distribution. It matters little whether the suggestion has come from the doctor or from the patient, the extent of the pain or loss of sensation demonstrably follows mental lines. The hysterical state is one in which such suggestion has unlimited effect.

When I say the word "hand," those of you who can visualise at once see a picture of the whole hand, front and back, limited by a line drawn round the wrist; in the same way the word "foot" calls up a picture of the dorsum and plantar aspects limited by a line at the level of the malleoli. In our minds these pictures represent the hand and the foot. If, therefore, the patient is in an over-suggestible condition and anything such as an injury calls his attention to his hand or his foot, it is over the extent occupied by these mental pictures that sensation will be disturbed.

In the same way the "belly" calls up a picture that stands in no relation to the "back"; hence in hysteria, sensory disturbances may occur on the one part without affection of the other, although they are anatomically and physiologically but the anterior and posterior aspects of a cylinder.

Thus, the face may be affected without the eye, lips or cheek; the spinal column without the back; the penis may be insensitive but not the meatus. In every case, whether the sensory change takes the form of pain or defective sensibility, its distribution is conceptual and not functional or anatomical.

Moreover, the quality of the sensory changes shows their mental origin. If a patient suffers from long-continued referred pains of visceral origin, the surface of the body will probably be tender. Pinching up the skin or dragging the point of a pin across the sensitive area will evoke discomfort. But if the place is lightly touched with cotton-wool, the patient will not complain because the stimulus contains no potentially painful element; pinching or dragging a point across the skin are potentially painful, for they have only to be carried out roughly to cause discomfort, even over normal parts.

But to the hysterical it is a particular part which is painful; the nature of the stimulus is indifferent provided it falls within the prohibited area. Thus, after marking out an abdominal area in the patient before you by dragging a pin across the surface, you see that I obtain as great or an even greater reaction by stimulating it with cotton-wool. In the same way, patients who complain of a tender spine resent gentle stroking up and down the centre of the back with cotton-wool—a stimulus that cannot evoke discomfort over this area in organic disease.

In the same way, the patient may contend that she is unable to recognise the position in space of the affected arm; in the case of the woman I am bringing before you this happens to be her right upper extremity. I close her eyes and tell her to find the right hand "with your good hand:" this she does at once. But ordered to find the left hand "with your bad hand," she waves it aimlessly in the air, and comes nowhere near the normal index she is supposed to be seeking. This is exactly the opposite to what would occur if there was a cortical lesion affecting the right hand; for then the "bad hand," making for a known goal, would make a better record than the "good hand" seeking a part the position of which in space was unknown. But to the hysterical the "bad hand" must of necessity make a worse record than the "good hand."

Similarly, if you ask this patient to kick out her "good leg," she does so easily, although it involves her supporting herself on the affected limb; but command her to kick out the "bad leg," and she falls over at once.

The paralysis of sensation or motion is conceptual: it is the direct fruit of an idea of disability.

In these lectures I have attempted to show you how the facts of sensory conductions may aid you in the localisation of disease. Practical points in clinical diagnosis are the bye-products of scientific knowledge, and nowhere is this better seen than in following up the diagnostic significance of sensory changes in diseases of the nervous system.

April 14th, 1913.

ACQUIRED MENTAL DEFECT.*

By A. F. TREDGOLD, L.R.C.P.Lond., M.R.C.S.Eng.,
Consulting Physician to the National Association for the Feeble-minded, etc.

GENTLEMEN,—Persons suffering from mental defect may be divided into two groups. In the first, which embraces between 80 and 90 per cent. of all cases, the defect is intrinsic—that is to say, it is due to an inherent blight, a deficiency or pathological change in the neuronic determinant of the germ-plasm, in consequence of which the cortical cells concerned with the manifestations of mind are unable to attain complete development, so that there results a varying degree of amentia. This group has usually been described as "congenital"; but, although the condition is inborn, it is not necessarily manifest at birth; it may not be apparent until several years after birth, and hence I think the better term is that of *intrinsic* or *primary* mental defect.

In the second group there is no morbid heredity and the germ-plasm is healthy; but the development of the cortical cells of the brain has been interfered with by some external influence, some factor of the environment. The mental defect which results is thus accidental or acquired, but since it may be acquired before as well as after birth I prefer to use the term "extrinsic" or "secondary" for such cases. It is with this group of *acquired, extrinsic* or *secondary* mental defect that I here propose to deal.

As I have just stated, the group is a relatively small one, probably comprising not more than between 10 and 20 per cent. of all cases of amentia; but amentia is so common that even this proportion means a very considerable number of patients. Moreover, the possibility of amelioration is so dependent upon early diagnosis as to make the group one of great importance as well as of no little interest to the practitioner.

The external factors which may in this way produce arrest of mental development are very numerous, and the pathological conditions found in the brain after death are of the most diverse nature. Nevertheless it is possible to divide the group into certain classes, and I shall acccordingly consider acquired mental defect under three headings, namely (1) toxic, (2) vascular, and (3) nutritional.

* A Lecture delivered at the Polyclinic.

CLASS I. AMENTIA DUE TO TOXIC CAUSES.

The class I shall first consider is that in which the arrest is produced by toxic agencies. As you are well aware, there are many substances which act as poisons upon certain of the body-cells. For instance, there is the inorganic group, well-known examples of which are arsenic, lead and phosphorus. There is the organic group, familiar instances of which are alcohol, carbon-monoxide, hydrocyanic acid, etc. Lastly, there is the group of toxins produced by the action of micro-organisms. It is these last which we find to be chiefly concerned in the causation of mental defect.

In most cases they would appear to bring about a real poisoning and necrosis of the cortical cells, but inflammation is generally present as well, so that the clinical signs of the process are those of an acute encephalitis, meningitis or meningo-encephalitis.

There are many micro-organisms which may act upon the brain cells in this way. For instance, the pneumococcus, gonococcus, various streptococci and staphylococci and the bacillus of influenza are all capable of causing cerebral inflammation and necrosis of cortical cells.

In such cases the affection of the brain is usually secondary to some acute process elsewhere, such as inflammation of the lungs, influenza, otitis, rhinitis, etc. More often, however, the affection of the nervous system would appear to be a primary one and to occur independently of lesions elsewhere, and then it is probably either a cerebral or cerebro-spinal meningitis caused by the Diplococcus intracellularis of Weichselbaum, or, as described by Strümpell, it is a polio-encephalitis analogous to the acute inflammation of the anterior horns of the spinal cord occurring in children. I am inclined to think that many cases of meningitis of obscure origin and many cases of so-called "sunstroke" and even "fright" really belong to one or other of these two last groups.

The clinical course of the illness is variable. In a proportion of cases prodromal symptoms are present, the child being listless and suffering from malaise for two or three days before any cerebral symptoms make their appearance. In most cases,

however, the onset is much more rapid, and he is suddenly seized with headache, vomiting and fever, speedily followed by convulsions or unconsciousness. The pain in the head is usually a marked feature, being very severe; pain in the back also is often complained of. The respiration is apt to be slow and irregular: the pulse may be slow or rapid, but is usually weak. The convulsions are general and resemble those of epilepsy; sometimes they are continuous so that a *status epilepticus* results; but in other cases they occur as isolated fits, the patient being either delirious or comatose between whiles. The symptoms follow no definite course and differ considerably in different patients, but they are always such as to make it quite obvious that the child is seriously ill, and that the seat of the trouble is the brain or its meninges. These symptoms are practically the same whether the brain is affected primarily, or whether it is involved in the course of some other condition, such as influenza or pneumonia.

In a few, but I think a very few cases, the child recovers completely. In others, after an illness of ten or fourteen days there is a marked change for the worse; the pupils dilate, the pulse and respiration become more irregular, the coma deepens and the patient dies. Sometimes the end comes after a series of convulsions. But neither of these terminations concerns us. There is a third result, and these are the cases to which I wish to draw your attention. The patient's life is spared, but the illness has left an indelible mark upon the brain cortex, with the consequence that he suffers from some degree of mental defect.

In most cases where the brain tissue has been thus damaged, the mental change is noticed at once. If the child had begun to speak he may now be speechless. His disposition may be altered, so that the persons, the playmates and the games of which he was formerly fond are now distasteful to him. With this there is either unusual irritability or apathy and dulness, and examination speedily shows the presence of intellectual impairment. As time goes on this is seen more and more in the difficulty he experiences in learning. In a few cases recovery from the illness at first appears to be complete, at any rate to the parents, and it is only after a time, when the child begins to go to school, that he is found to be incapable of mental application and of progressing with his studies. Incurable deafness is not uncommon in cases of this kind. With regard to the degree of defect the brain lesion may be so severe that the child is rendered idiotic from the beginning; in other instances it is much less serious, but the patient cannot keep abreast of his compeers, and as time goes on he is left further and further behind in a state of incurable feeble-mindedness. I cannot say what proportion of children who have suffered from encephalitis or meningitis are thus rendered permanently weak-minded. No doubt, as I have remarked, very many of them die, whilst a few seem to recover completely; but the likelihood of interference with brain development is so great that the utmost caution should be exercised in giving a prognosis. It will be more convenient to discuss the factors influencing our opinion after we have considered the next class.

Class II. Amentia due to Vascular Lesions.

In addition to the arrest of cortical development brought about by toxins, cerebral lesions may be produced and growth interfered with by such vascular changes as hæmorrhage, thrombosis and embolus. It is this class we have now to consider.

The commonest of these lesions is undoubtedly hæmorrhage. Thrombosis occasionally results from asphyxia neonatorum, and embolus may occur in young infants suffering from rheumatic endocarditis, but both of these are comparatively rare. The hæmorrhage is generally meningeal and usually takes place at the time of birth, being caused by the prolonged pressure incident to a protracted labour. The forceps have often been blamed for the bleeding, but it is very doubtful to my mind whether skilfully applied forceps have ever of themselves produced serious injury to the brain. Rupture of a cerebral blood-vessel, too, may take place during a severe paroxysm of whooping-cough, and at times it follows direct trauma in the shape of a fall or blow. This last may occur before as well as after birth, and may be accompanied by laceration of the brain cortex.

In the great majority of the birth cases, which is by far the most numerous group, the child presents well marked asphyxia when born, from which he is resuscitated with considerable difficulty. For several days he remains torpid, with feeble pulse and respiration, tense anterior fontanelle, and contracted pupils. He does not cry, and requires

much rousing before he will take food. His muscles are rigid, opisthotonos may be present, and convulsions are frequently seen. A little later on evidence of paralysis in one or more limbs is noticed. Sitting up, walking and speech are all delayed, and it is soon apparent that the child's mind is not developing as it should. As time advances the fact that his intellect is impaired becomes more and more obvious.

Paralysis is a very common feature of these cases, and the condition is often termed "paralytic amentia." It may be a monoplegia, hemiplegia, diplegia, or paraplegia. Probably diplegia and paraplegia are really the most frequent lesions, but, since these mean a very severe affection of the brain, the majority of such die within the first few weeks or months of life, and hence it is a monoplegia, or hemiplegia, that we usually see in association with this variety of mental defect. In addition to being paralysed, the affected limb remains livid, cold, and much smaller than the corresponding healthy one. Jacksonian or epileptic convulsions occur in a considerable proportion of these cases, and athetoid or choreiform movements are very common. There is usually some flattening of the skull over the affected side. The degree of amentia varies from gross idiocy to mild feeble-mindedness.

These two, the toxic and the vascular, are the commonest varieties of secondary or acquired amentia, and to one or other of them you will be able to refer the great majority of cases of mental defect which are not of intrinsic origin. I have dealt with them separately up to this point because it seems to me important that we should understand their different modes of origin, but we may now consider a few facts applicable to both of them. And first of all it is worth bearing in mind that the lesions seen upon the post-mortem table long after the incidence of the initial pathological process generally differ very considerably from those at the beginning. We do not yet know all the factors bringing them about, but it is quite certain that many secondary changes may result, so that in one case we find areas of sclerosis, with shrinkage, in another chronic meningitis, cortical agenesis and heterotopia, cysts, porencephaly, hydrocephaly, or the like. Sometimes these give rise to more or less distinct types which may be recognised clinically, but it must be remembered that these lesions are only after-effects, and that the real cause of the defect is the destruction of a cortical area, to which may be added an arrested growth of those parts of the brain which are anatomically connected.

With regard to the diagnosis of these cases, if several months or years have elapsed since the incidence of the primary lesion there will seldom be any difficulty. If real defect is present it will readily be ascertained by a psychological examination on the lines I have indicated in a previous lecture, and that the defect is of the secondary variety will be shown by the absence of any neuropathic heredity and by the presence of a history of a toxic or vascular lesion like those I have described. The mental defect may be of any degree, and although idiocy usually reveals itself by its characteristic physiognomy, it is well to remember that those patients who are merely feeble-minded or even imbecile may have the outward appearance of ordinary individuals. This arises from the fact that the germinal material is healthy, the defect is purely accidental, and, consequently, is unaccompanied by any of those anomalies of bodily development—stigmata of degeneracy—which are such a distinctive feature in cases of primary amentia. Persons suffering from acquired mental defect are, indeed, as remarked by Langdon Down, often "well grown, comely and winsome."

If the case is seen at the onset the matter is very different, and you will often find it exceedingly difficult to answer the parents' anxious inquiry as to the likelihood of intellectual impairment resulting.

Of the children born with asphyxia, the number in whom amentia results is extremely small, and careful observation of the child for a few days will usually enable the physician to reassure the parents on this head; unfavourable symptoms are convulsions, extreme torpor or persistent screaming. Of the toxic cases happening during early childhood, the proportion who become aments is much larger, and this possibility can never be excluded with certainty until the lapse of some time after the illness. It is very fallacious to judge of the probability of mental impairment from the presence of paralysis, whatever the nature of the lesion. Should paraplegia or diplegia be present it is true that there will probably be some degree of incurable mental defect, because this means that both hemispheres have been

involved, and it is obvious that the greater the extent of the lesion the more likelihood will there be of psychic injury. But, apart from this, the mere presence of paralysis is a very uncertain indication. I have seen many instances of severe hemiplegia in which the children grew up, not merely non-defective, but of unusual brightness and intelligence; conversely I have seen profound secondary amenta, caused by a birth injury or toxic infection, without any trace of paralysis. No doubt these differences have been due to the particular cortical region which has been involved by the morbid process.

The symptom of most unfavourable import is undoubtedly epilepsy, and if convulsions continue, even at comparatively rare intervals, after the fever has subsided and the child has recovered from his original comatose condition, then the mental outlook is very serious. I do not mean to say that in every case which is followed by occasional epileptic fits, even if some paralysis be present as well, the prognosis as regards improvement is necessarily hopeless. This is not so, but it is an undoubted fact that in the larger proportion of the cases in which epileptic convulsions occur mental impairment will result, and only too often to a severe degree.

This leads me to say a few words regarding *epileptic amentia.* In the cases I have just referred to both the amentia and the convulsions are the result of a common cause; they are, in fact, symptomatic of, and produced by, a gross cerebral lesion. It often happens that at first the fits are Jacksonian in character; later on, however, they tend to become generalised and to resemble those of ordinary idiopathic epilepsy. But idiopathic epilepsy, that is, epilepsy occurring in the absence of any demonstrable morbid anatomical condition, may itself give rise to mental defect, and it is to defect so caused that the term "epileptic amentia" is applied. You are well aware that repeated epileptic attacks, whether of the minor or major variety, are very liable to degrade the intellect and produce some degree of dementia; consequently you will readily understand that should these attacks occur at an age when mental development is still incomplete, they will be very liable to arrest that development and so produce secondary or acquired mental defect.

If epilepsy occurs before the age of five years, the probability of development being thus arrested and some degree of defect being produced is very great. Much, however, depends upon the frequency of the fits and their response to treatment. I have seen several cases where marked arrest was present, but where the successful treatment of the epilepsy was followed by a considerable improvement in the mental status. I may here remark that it is sometimes stated that mental defect may follow "fright" or "shock." Undoubtedly such causes may bring about arrest in those hereditarily predisposed, but I doubt whether they are ever capable of so acting in a normal child.

Before leaving these two varieties of defect, let me add a few words regarding their treatment. Speaking generally, it may be stated that if the case is really a secondary one, and not a case of primary amentia complicated by a gross cerebral lesion, there is a probability of a fair amount of improvement under proper educational methods. And on the whole I am inclined to think that the prospects of amelioration are better in lesions of purely vascular than in those of toxic origin. This may partly be due to the fact that vascular lesions generally occur during birth or at an earlier age than those of a toxic nature; but it may also be due to some extent to the difference in the pathological process.

It might perhaps be thought that benefit would result from some surgical procedure, and as a matter of fact parents are often very anxious for the performance of an operation. I must confess that I have very little experience of the effect of operation in such cases; possibly if one could diagnose the presence of a blood-clot and locate its position operation might be undertaken, but in other cases it is difficult to see how this would possibly be of any benefit, and I should certainly be very loth to recommend it.

Whatever the nature of the morbid process, its effect is to destroy or arrest the development of a group of cortical cells and their ramifications, and treatment must be directed firstly, towards limiting the lesion, and secondly, towards educating other cells to perform the work of those which have been rendered *hors de combat.* With regard to the first of these desiderata, I doubt whether drugs are of much use. I have frequently prescribed iodide of potassium, also mercury, in view of their supposed resolvent action, but I much

doubt whether they have had any real effect. More can be done, I believe, by careful attention at the first to avoid undue light, noise, movement, or any external stimulus which might increase cortical excitement. The condition of the bowels requires attention, and when recovery is taking place abundant sleep is essential; it should be obtained by paraldehyde or the bromides in preference to opium. A little later plain nutritious feeding is needed, and I think that iron, arsenic and the glycero-phosphates sometimes prove of value in assisting cerebral nutrition; strychnine must be given with the greatest possible caution. Provided the lesion has happened whilst the cortical cells are still in an early stage of structural development, there is, to my mind, some evidence that compensation may occur through the development of other neuroblasts to take the place of those which have been destroyed, and this supplies the indication for our chief treatment, namely, regular systematic training. I do not propose to go into the details of this; it can only be carried out properly by a skilled, patient teacher, acting under the constant supervision of an expert who is capable of forming an opinion as to the nature of the psychic injury and the most appropriate methods of remedying it. This much, however, is certain—that training of this kind is absolutely necessary if improvement is to result, and that it must be begun at the earliest possible moment. No more fatal plan can be adopted than the, I am sorry to say, only too common one, of deluding the parents with the fond hope that all will come right with the lapse of time, and that the child will grow out of it "when he is seven."

Class III. Amentia due to Nutritional Defect.

The last group of cases to which I wish to direct your attention are those in which the normal growth and development of the brain are defective in consequence of inadequate nutrition. Like every other organ in the body, the growth of the brain is dependent upon two influences; firstly, a tendency inherent in a certain portion of the fertilised germ-cell to grow, to proliferate and to become differentiated into various orders of nerve-cells, each having its own particular connection and peculiar function; secondly, upon the presence of a nutritive material adequate in quality and amount.

Now it must be confessed that at present we know very little regarding the nutritive requirements of the various bodily organs, but it seems certain that the brain, at any rate, requires for its growth certain particular chemical constituents, and, if these are lacking, then normal development cannot take place. We then have a form of amentia which I have called "nutritional."

One of the most interesting and best known examples of this variety of defect is *cretinism*, which is due to an inadequacy of the secretion of the thyroid gland. The cause of the athyroidea we do not yet know, neither do we know whether it is some particular constituent or the whole of the secretion which is necessary; but that the brain-cells cannot attain full development if this secretion is wanting is quite clear; consequently all cretins suffer from some degree of mental defect. We see a similar effect upon cerebral nutrition in the myxœdema occurring in later life. The signs and symptoms of cretinism are so well known to you that no description is necessary here I merely mention it as a very interesting example of the group of acquired mental defects due to defective nutrition.

There is another very interesting type of amentia known as *Mongolism*, which is often mistaken for cretinism, although a close examination will readily show the difference. The pathology of this condition is very obscure: heredity seems to play some part, although perhaps a slight one, and in most cases there are indications of exhaustion on the part of the maternal generative organs. It may eventually be found that here also there is a deficiency of some internal secretion which is vital to the development of the brain.

Leaving these specific deficiencies let us now consider the question of the effect of general bodily malnutrition upon mental growth. The statement is commonly made that the defective nutrition and the insanitary surroundings which *inter alia* form part of "slum life" are responsible for mental defect. So far as my experience goes this is very rarely the case. I have seen instances, it is true, where the normal evolution of the mental faculties has been considerably in arrears in consequence of such conditions. I have seen a few cases where these arrears have never been made up, so that some degree of real amentia has resulted. But such cases are relatively few, and if you can exclude the presence of any hereditary tendency you may reassure the parents, for as the bodily condition improves under good feeding, sunlight and fresh air, combined, if necessary, with suitable medicaments, it is extremely probable that the mind will undergo a like change.

April 14th, 1913.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from page* 16.)

(187)

Syphilis no Respecter of Persons, or Medical Men and Morals.

By not recognising that syphilis may be the cause of a headache, the practitioner often loses his opportunity for early treatment, with possibly disastrous results to the patient. I cannot too strongly insist, even by repetition *ad nauseam*, on the importance of this matter, nor on the fact, too, that the medical man must not allow the reputed respectability of the patient to stand in the way of the idea of syphilis. Over and over again I have had men say to me, especially in consultation practice, that the condition of a particular patient could not be syphilis because the patient had been coming to him for years and was utterly respectable and his reputation was unblemished, just as if syphilis was something closely bordering on criminality, something very sinful in itself. Anyone may have syphilis, and if a person has a disease he must be treated for that disease, whatever his reputation may be. Remember it is nothing to do with the doctor; it is not the doctor's duty to investigate reputations; he has to investigate and treat disease. In a recent case the Lord Chief Justice pointed out that a court of law was not a court of morals. Clergymen and priests seem to be ambitious to treat disease, but that, of course, is not their function—*ne sutor*. It is well to understand that syphilis is no more sinful than measles, typhoid fever, or smallpox, and to remember that it is of vital importance to the community that syphilis should be treated quickly and adequately.

("Syphilitic Headaches," by George Pernet, M.D., 'Clin. Journ.,' vol. xxxvii, p. 81.)

(188)

Nocturnal Headache and Syphilis.

When patients tell you they have no headache during the day, but that it comes on at a definite time in the night, that should make you suspicious that syphilis is at the bottom of the trouble; certainly you should investigate the case carefully from that point of view.

(*Ibid.*)

(189)

The Confidence of Youth.

The tube is left out by some wiser and younger mind, septic osteitis ensues, and in time the neighbouring joint becomes involved.

("The Treatment of Central Osteitis by Metal Drains," by Charters J. Symonds, M.S., F.R.C.S., 'Clin. Journ.,' vol. xxxvii, p. 85.)

(190)

Impartial weighing of all Evidence necessary for Correct Diagnosis.

I want you to let this case (an obscure case of colic) remind you that rational treatment must be based upon accurate diagnosis, and that accurate diagnosis can only be made by carefully weighing all the available evidence, without paying too much attention to any part of it, however fascinating that may be.

("The Importance of Thorough and Rational Examination of the Urinary Organs," by R. P. Rowlands, M.S., F.R.C.S., 'Clin. Journ.,' vol. xxxiii, p. 295.)

(191)

Nervous Disease as a Cause of Retention.

Disease of the central nervous system must be borne in mind as one of the causes of retention. Prostatectomy has been performed under the impression that the patient was suffering from prostatic retention, and the fact that it was tabetic in origin entirely overlooked. Needless to say the patient's condition was not improved.

("Retention of Urine," by Hugh Lett, M.B., F.R.C.S., 'Clin. Journ.,' vol. xxxvi, p. 281.)

(192)

"Nipping," not confined to Alcohol.

Many women who are really invalids, or approaching the stages of invalidism, have a tendency to be always eating, to be frequently taking food between their ordinary meals, and especially numerous patent preparations. One constantly sees patients whose acquaintance with all the patent foods and liquids is astounding. You will frequently find that the stoppage of them will do your patient a great deal more good than by any treatment of the uterus.

("Gynæcological Therapeutics," by Frederick McCann, M.D.Edin., F.R.C.S.Eng., 'Clin. Journ.,' vol. xxxvii, p. 113.)

(193)

Why Men and Women drink.

It has been said that men drink from feelings of conviviality, women because of physical suffering.

(*Ibid.*)

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THE CONTRACTION RING AS A CAUSE OF DYSTOCIA.*

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DYSTOCIA from the formation of a contraction ring over a depression in the child's outline is probably a moderately common complication of labour. It is, however, not usually recognised as such, and the ring itself is mistaken for the retraction ring of Bandl, which is a result of labour obstructed by some other cause, in fact the cases which are recorded in the literature are frequently described as due to a "retraction" ring instead of a "contraction" ring. As the two conditions are quite different, and I have recently had the opportunity of examining a uterus with a contraction ring removed during labour, I have attempted to give the differential diagnosis. A correct diagnosis in these cases is of the utmost importance, for, as will be seen, the treatment is difficult and the mortality high.

This differentiation of the two conditions is only tentative, for as further knowledge is gained, so the question of classification will have to be reconsidered. Probably under the term "contraction ring cases" several clinical entities are included which differ from each other in important points, for instance in the amount of relaxation of the body of the uterus with its consequent effect on the presenting part.

DIFFERENTIAL DIAGNOSIS.

Contraction ring.	Retraction ring.
1. A localised thickening of the wall of the uterus due to the contraction of the circular fibres over a point of slight resistance, most frequently over a depression in the child's outline or below the presenting part.	The junction of the thinned lower uterine segment with the thick retracted upper uterine segment.

* Read at the Royal Society of Medicine.

Contraction ring.	Retraction ring.
2. The uterine wall at the site of the contraction ring will therefore be thicker than it is either above or below.	The uterine wall above the retraction ring is much thicker than below it.
3. The wall below is neither thinned nor over-distended.	The wall below a retraction ring is both thinned and over-distended.
4. The presenting part is not forcibly driven into the pelvis.	The presenting part is, or has been, jammed into the pelvis.
5. The child may be wholly or mainly above the contraction ring.	Part of the child must be below the retraction ring.
6. The body of the uterus above a contraction ring is usually relaxed and not tender.	The body above a retraction ring is tonically contracted and hard.
7. Round ligaments are not tense.	Round ligaments stand out.
8. A contraction ring may occur in the first, second or third stage of labour.	A retraction ring practically always occurs late in the second stage of labour.
9. A contraction ring does not vary in position as labour goes on.	A retraction ring gradually rises as retraction of the upper uterine segment proceeds.
10. A contraction ring is rarely felt on abdominal examination.	A retraction ring may frequently be felt *per* abdomen.
11. The patient's general condition is good.	The patient's general condition is bad.
12. Causation: Premature rupture of the membranes. Intra-uterine manipulations.	Causation: Obstructed labour.

(1) The Existence of a Local Contraction of the Uterus.

Herman (26) and Veit (45) seem to doubt the existence of a localised contraction of the uterus apart from obstructed labour, and yet there is nothing inherently improbable in its existence, as Bar (3) noted that the two tubes of the double uterus of a rabbit need not contract simultaneously, and Treub (43) showed the same thing in a double human uterus. The actual existence of such a contraction ring is proved by measuring the thickness of the wall of a uterus, and noting that in the ring it is thicker than above or below. This was done in one of Philipps's cases, recorded by Holland (28), in Andrews and Maxwell's (1), Schroeder's (38), Lusk's (32), and the specimen shown to-night. Berry Hart (25) pointed out that during labour the anterior wall of the uterus was about 6 mm. thicker than the posterior, so that it is essential that all measurements should be taken on the same side.

(2) The Ring forms over an Area of Slight Resistance and tends to fit into Depressions on the Child.

In Andrews' and Maxwell's case the ring corresponded with the groove of the child's thigh, in one of Budin's (10) to the hollow of the knee. Forming below the feet in incomplete breech presentations, its importance is insisted on by Lewis (31), Jardine, and de Bovis (7). It has also formed round the thorax (Chavane (12), Boquel (6)), shoulder and neck (Demelin (15) and others), and round the arm (Bué (11), Hicks (27), and others), but in the great majority of cases it forms round the neck. In two cases (Budin's and Fabre and Dangat's (20)) the fœtus was anencephalic. Finally, it may form below the presenting part (Smith (40), Sinnetamby (39), Jardine, Formichella (21) and others).

The position of the contraction ring in the cases I have collected is: Round neck, about 45 per cent.; below the whole of the child, 21 per cent.; round the arm, 15 per cent.; round thorax, 9 per cent.; neck of after-coming head, 9 per cent.

In showing the part played by the fœtal outline in the formation of the ring, an observation of de Bovis is of importance. The ring fitted tightly into the popliteal space of a child presenting as an incomplete breech. He dilated it with difficulty and finally reached a foot, but the prominence of the ring prevented the foot being brought down. On following the ring backwards to the level region of the child's back, he states that he was agreeably surprised to find that the ring became less and less marked, till it was practically non-existent. He took advantage of this in finally delivering the child.

(3) The Wall below the Contraction Ring is not Thinned or Over-distended and the Presenting Part not Forced Down into the Pelvis.

In a retraction ring case the thickening of the upper uterine segment is compensated for by a thinning of the lower segment, and at the junction of the two the ring forms. The presenting part

must have been fixed in the pelvis in order to give the uterus a point *d'appui* to contract and retract against; for instance, it is inconceivable that thinning of the lower uterine segment could occur with a hydatidiform mole. The presenting part is jammed and fixed in the pelvis, except in the theoretical case where secondary uterine inertia has supervened on the top of tonic contraction.

On the other hand, with a contraction ring the wall below is not thinned, neither is it over-distended. Clinically, thinning is difficult to determine, and so more importance should be paid to the absence of distension than to apparent thinning. Demelin (17) describes the wall below as "*flasqué et lache*," Dickinson (18) as relaxed and not thinned, and Sinnetamby as hanging loosely in the pelvis below the ring.

The presenting part is, moreover, as a rule, not fixed or forced down in the pelvis. In some cases it is below the presenting part—this alone should exclude a retraction ring at once. When round the neck the head is not fixed, and this (as shown experimentally by Demelin) is one of the reasons why forceps fail, as they slip off unless applied over the occiput and face. Spiegelberg (41) gives the clinical picture when describing what he calls "close application of the uterine wall to the fœtus." He says the condition is generally called "tetanic constriction," but he continues, "Such is not the true condition, for the presenting part is not firmly impacted in the pelvic brim, the uterus is not sensitive to pressure, nor does the hand, on being introduced, meet with any great resistance."

Condition of the Body of the Uterus.

In a contraction ring case the fundus, as a rule, is soft and still undergoing contractions and relaxations. In a case of Brindeau's (9) the body was so soft that the presentation of the child changed after rupture of the membranes, and in several cases (Sinnetamby, Radcliffe (35)) *post-partum* hæmorrhage occurred, which is very rare in tonic contraction. The ring, when sufficiently marked to cause dystocia, does not relax with the rest of the uterus. Budin even states that it seemed to get tighter. In exceptional cases the body of the uterus is hard and does not relax. In the same way the fundus, as a rule, is not tender.

Time of Occurrence.

Except in rare cases of organic obstruction to dilatation of the cervix (*e.g.* by carcinoma) a retraction ring is always seen late in the second stage.

Contraction rings are also most frequently found in the second stage, probably because it is at this time that the hand is introduced to find the cause of the delay, but they are met with in the first stage with the membranes still intact (Budin, Smith, Jardine and Demelin (17)). The occurrence of a contraction ring during labour is followed by retention of the placenta in some 7 per cent. of cases. In Chavane's case the placenta was retained for twenty-four hours: he then called in Maygrier, who had the greatest difficulty in getting through the ring. During the puerperium intra-uterine douches were necessary, and he records that even the intra-uterine tube was gripped, thus showing the extreme irritability of the uterus. Hahl (24) noted gripping of the sound 6 cm. from the external os two months after delivery, when involution was so far complete that the total length of the uterine cavity was 9 cm. In a case recorded by Perret (34) the ring again contracted tightly round his wrist while removing the placenta manually, and yet the fundus was sufficiently soft for him to proceed with the separation of the placenta although the wrist was being tightly held. Dystocia by a ring has also occurred in three successive labours in the same patient (Smith), and has been noted with the second of twins (Smith, Williamson), and the last of triplets (Smith).

Abdominal Palpation as a Means of Differential Diagnosis.

Several writers state that the retraction ring is more easily palpable *per abdomen* than a contraction ring, but in a large number of cases of obstructed labour, owing to obesity, rigidity and other causes, the ring is difficult to feel, so that obstetricians of large experience may never have felt a retraction ring by abdominal palpation; on the other hand, the contraction ring has been felt on abdominal examination (Dickinson's third case, Hicks's first case). Thus it appears very doubtful if this will be of any practical help in making a differential diagnosis.

General Condition of the Patient.

When seen early, before being exhausted by fruitless obstetric operations, the patient with a

contraction ring is in good condition compared with the exhausted state of a patient with obstructed labour. For example, one of my cases after four days in labour had a pulse of 84 and was comfortable. Hicks describes a case where the patient was free from pain although the ring was so tight that cephalotripsy failed.

Causation.

(A) *Premature rupture of the membranes.*—This is the most constant factor in records of cases.

(B) *Intra-uterine manipulation.*—These cases are also very frequent, but it must be noted that unless some intra-uterine manipulation had been done, the diagnosis could not have been made. The use of hydrostatic bags has been alleged (14) to cause the condition, but is unimportant. When used in cases of placenta prævia the abnormal condition of the lower segment produced by the low insertion of the placenta is probably of more importance than the bag.

(C) *Increased irritability of the uterus.*—It is obviously necessary to presuppose the existence of increased uterine irritability in addition to the two factors mentioned to explain the infrequent occurrence of contraction rings.

(D) *Malpresentations.*—All forms are common. Cheron's (13) statistics are: Vertex, 62 per cent.; shoulder, 21·4 per cent.; face, 7·0 per cent.; breech, 8·6 per cent. De Bovis and Jardine lay stress on the frequency of incomplete breech presentations.

(E) *Maceration of the fœtus and infection of the uterine contents.*—Zweifel (49) drew attention to this in reporting a case. About 10 per cent. of recorded cases have been delivered of macerated children. Regarding the question of intra-uterine sepsis, the death-rate from peritonitis in cases treated by conservative Cæsarian section is high enough to show that it must be common.

(F) *Uterine inertia.*—A long first stage with weak pains is usual in these cases. Blacker (5) lays stress on the association of uterine inertia with trismus of the cervix. Gilles (23), in recording a typical case, draws attention to the extreme inertia which was present for four days and persisted till the ring was divided during Cæsarean section.

(G) *Parity.*—Dickinson states that primiparæ and multiparæ are equally affected in his series of ninety cases. The cases which I have found in the literature described under various names amount to about the same number and overlap Dickinson's series very little; in my cases it is twice as common in multiparæ as in primiparæ.

Prognosis.

This is bad for the mother and worse for the child. In Smith's series, which included several obvious retraction ring cases, the maternal and fœtal mortality was 27 per cent. and 83 per cent. Dickinson concludes that in well-marked cases one third of the mothers and one half of the children die. The present series (excluding laparotomy cases) shows a mortality of 38 per cent. and 63 per cent. In nineteen cases treated by laparotomy (excluding one death from eclampsia) the mortality is 31·5 and 42 per cent.

Three cases of labour with this complication have come under my observation.

The first patient was æt. 38 years, and had had two previous children (one normal delivery and one forceps). Labour started about mid-day on May 18th, 1907; the membranes ruptured at 10 p.m. and high forceps were applied by her doctor, but failed. At 1 a.m. version was attempted, but only one leg could be brought down as far as the brim. At 3.30 a.m. she was admitted to Queen Charlotte's Hospital under the care of Dr. Arthur Stabb. Pulse 128, temperature 97·4° F. General condition good. The uterus was relaxed, and like a case of secondary uterine inertia; the fœtal parts could be easily palpated. No ring felt on abdominal examination, but on vaginal examination a marked contraction ring could be felt. The child presented by the head and feet, the body being arched over the fundus. The whole child, including the head was above the ring. The ring ran round the uterus about two inches above the brim and prevented the head engaging. The fœtal parts were movable, but even pressure on the head applied through the abdominal wall could not bring the head down into the brim. I repeatedly attempted to deliver by craniotomy, but failed to get the head past the ring and finally ruptured the uterus through the posterior wall; the tear involved the ring. The child was now easily delivered by version, the placenta removed by hand, and the tear plugged with gauze. The patient died of shock shortly after delivery. No necropsy was obtained.

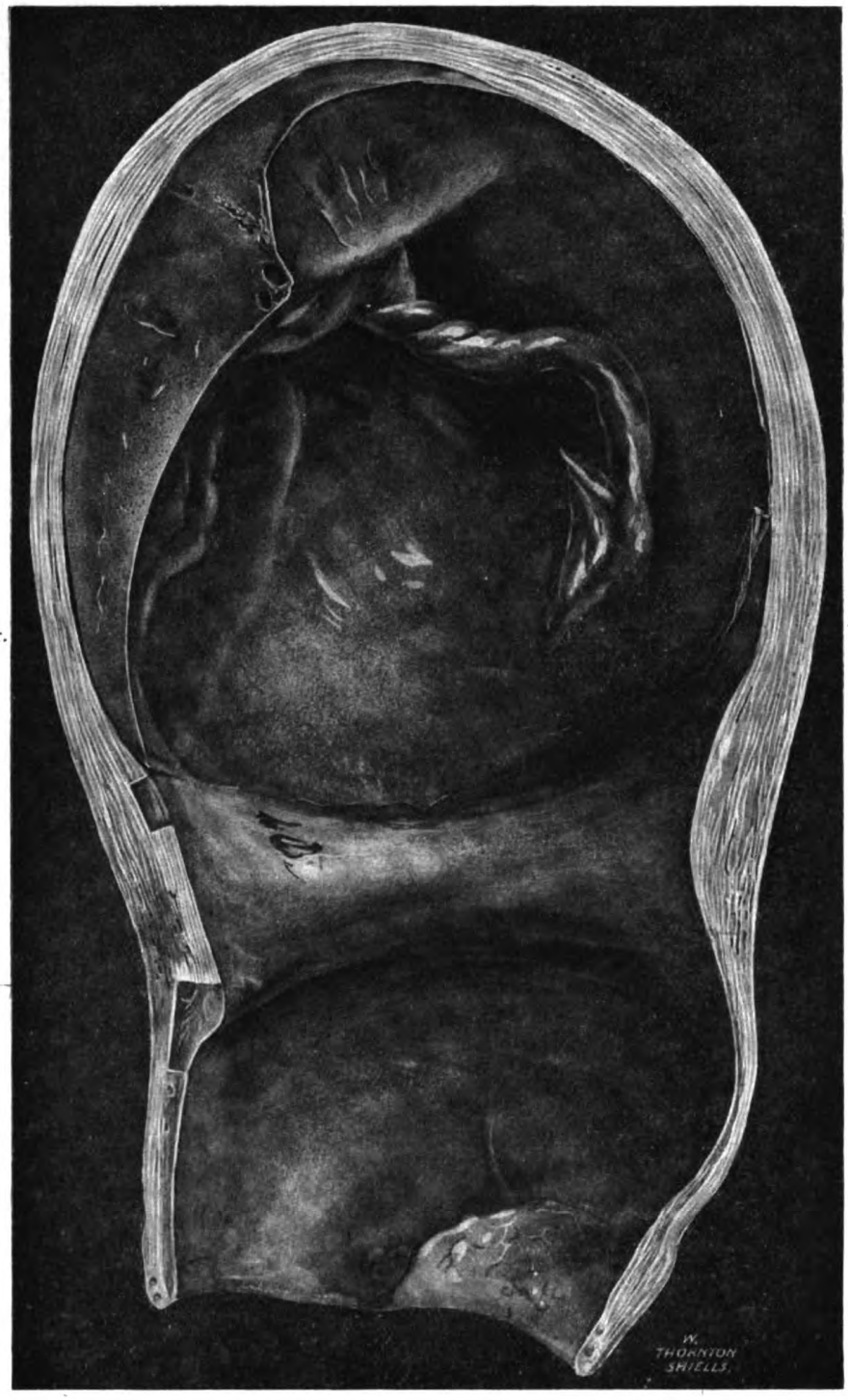
The second patient, æt. 31 years, had had five

children. She was admitted to Queen Charlotte's Hospital under my care on account of a placenta prævia on July 15th, 1912. The child presented by the vertex. At 8 p.m. the membranes were ruptured and a de Ribes' bag inserted. At 9.30 Dr. Gilliat, the senior Resident Medical Officer, reported that the bag had been expelled, but on attempting version he had found a contraction ring 2½ in. in diameter above the place where the bag had been, and 4 or 5 in. from the cervix. The placenta was partly below it, but the whole child was above it. The child being dead I advised craniotomy and cautious traction; this, together with cleidotomy, effected delivery. The third stage was normal. The child (after perforation) weighed 5¼ lb. The mother did well.

The third case is the one from whom the specimen shown to-night was removed. The history of the case is as follows: The patient, was æt. 34 years, and had had four normal labours. Her fifth labour started on July 31st, 1912; the membranes ruptured the next day and feeble pains continued till August 4th. Dr. James Wigg was then called and found an arm prolapsed; he brought down a leg, but failed to get the child to move as a whole. The contraction ring could be felt, but it was possible to get the hand beyond it. The patient was then sent to University College Hospital. On admission at 2 p.m. her pulse was 84, temperature 99·2° F. The uterus was contracted, but relaxed at intervals and was not tender. Fœtal heart not heard. Chloroform was given, and version was attempted by Mr. Twining, the Resident Obstetric Assistant, but was not persisted with when the contraction ring was felt. Morphia (⅓ gr.) given. In the absence of the Obstetric Physician, I saw the patient at 5 o'clock. Her general condition was very good: uterus contracted but not tender; the round ligaments were not standing out. Bandl's ring was not felt *per abdomen*. *Per vaginam* the lower uterine segment was not tense or thin, a foot and hand were presenting, but the presenting parts were not jammed down at all tightly into the brim. The cervix was fully dilated, so that the position of the external os could not be identified. A tight ring could be felt contracted round the arm; the lower level of the ring was so high that it could only be felt when the whole hand was introduced into the vagina. The ring was so tight that the finger could not be passed beyond it, and the use of any instrument for a destructive operation was out of the question. A culture was taken from the cervix and showed staphylococci. The patient had been torpid with morphia for two hours, and yet, even when chloroform was pushed to its full extent, no relaxation of the ring occurred. An attempt was made to give an intra-uterine douche as advised by Maxwell (33), but the ring was too tightly contracted on the arm to allow of the tube being passed to the fundus, the abdomen was then opened, and the uterus, with the fœtus inside, was removed entire. The ovaries were healthy, and were not removed. Before cutting through the vagina, curved clamps were put on to the cervix as far as the prolapsed arm would permit, in order to prevent the contents of the uterus from soiling the peritoneum. The convalescence was without incident. The specimen was at once put into formalin to harden; the hardening process was completed by injecting strong formalin into the tissues of the fœtus.

Description of the specimen. — The specimen shows the two halves of a full-term pregnant uterus divided into saggital section. The vagina has been cut through at its junction with the cervix. The child has been removed from the left half (Fig.); in the right half the fœtus is still *in situ*. The specimen measures 25 cm. in length. Its antero-posterior measurement is 15 cm. at its widest part and 9·5 cm. at its narrowest below. Externally no ridge or furrow is to be seen, but a ring can be seen running round the interior of the uterus at the junction of the lower and middle thirds. It runs obliquely so that in front it is 2 cm. higher than it is behind. The ring measures 3·5 cm. to 5 cm. from above down, and from 1·5 cm. to 1·7 cm. in thickness, this being an increase of 5 mm. to 8 mm. on the wall above, and 8 mm. to 10 mm. on the wall below. The fundus is 6 mm. thick. The ring is most sharply defined on the left side, where it constricted the child's neck (Fig.). The child is almost wholly above the ring, and is lying in a very contorted position; the head is in the left half of the uterus, midway between the cervix and the fundus, and immediately above the ring. The ring thus constricts the neck and thorax. The right arm is prolapsed into the vagina, the left is above the head. The left leg is prolapsed beside the right arm, the right foot is at the highest point of the uterus, the leg lying along

Posterior.



Anterior.

FIG.—Mesial saggital section of uterus removed during labour showing the contraction ring at the junction of the lower and middle thirds of the uterus. The ring corresponded to the depression of the child's neck, the head being immediately above the ring and the shoulder below it.

the anterior wall. The neck is deeply grooved where the ring had compressed it. The child is a male, and weighed (after hardening) 5¼ lb. The placenta lies on the posterior wall near the fundus and wholly above the ring. Microscopic sections were made of the wall of the uterus at various parts of the ring, but all histological detail had been destroyed by the strong hardening reagents that had been employed to harden the specimen.

Treatment of Dystocia by a Contraction Ring.

(1) *Expectant.*

This is useless, as in a severe case the condition persists. Budin waited thirty-six hours in one case but the ring did not relax.

(2) *Drugs.*

(A) *Anæsthetics.*—Acting on the supposition (probably incorrect) that the condition is one of pure spasm, both chloroform and ether have been tried very fully, but have entirely failed to relax the ring in severe cases. Vallois (44), when delivering with craniotomy forceps, found that the ring did relax, but, unfortunately, it appeared that the moment of delivery coincided with the death of the patient from (it is alleged) an overdose of chloroform.

(B) *Sedatives.*—Morphia has failed even in large doses. Chloral hydrate has not been given any extensive trial, but from its action in cases of trismus of the cervix might be of use.

(C) Isolated cases are recorded where other drugs have apparently done good. Souter (42) claims to have obtained, in two cases, a temporary relaxation of a tonic uterus which enabled version to be successfully performed, by administering 10 minims of amyl nitrite by inhalation. Williamson suggested atropine, but had not tried it. Hyoscine hypodermically failed in Watt's case (46). Hot baths are said to have caused relaxation in one of Budin's cases.

(3) *Dilatation of the Ring.*

(*a*) *Manual.*—In the slighter cases this frequently succeeds. Budin suggested leaving the hand *in situ* to give the head an inclined plane to roll up. In severe cases the fingers cannot effect dilatation.

(*b*) *Instrumental.*—Bowen (8) and Hahl tried these and failed.

(*c*) *Dilatation by traction* ("*Willett's continuous weight traction*").—This ingenious method is limited to cephalic or breech presentations. An instrument (*e. g.* craniotomy forceps) is fixed to the fœtus and a weight traction of 6½ lb. suspended. It succeeded well in his case.

(4) *Forceps.*

These are limited to cephalic presentations with the head below the ring. In the more severe cases they fail entirely. The head is suspended by the ring round the neck, and in the majority of cases as soon as traction is made they fall off. Demelin, from experiments made, advises their application over the occiput and face so as to avoid the movements at the occipito-atlantal joint, which causes them to slip off as soon as traction is made on the suspended head.

If a good grip is obtained, the usual effect of traction is to drag the uterus *as a whole* down towards the vulva. Rupture of the uterus from the use of forceps has been known (16).

(5) *Embryotomy.*

Where forceps and version have failed, the destructive instruments have, as a rule, been tried next, but all writers agree that the embryotomies have been extraordinarily difficult from the high position of the head and the difficulty of getting the instruments past the ring.

After perforation of the head, the shoulders are held back if the ring is round the neck, and traction by craniotomy forceps only moves the uterus as a whole. Cleidotomy is therefore indicated in every case where the ring is round the neck of a dead child.

Hicks cut off the head by curved scissors when cephalotripsy failed, and then delivered the remainder of the body by internal version above the ring. Andrews and Gaulaud (22) record cases where embryotomy has failed to deliver the patient.

Cæsarian Section.

Cæsarian section is indicated where the ring is wholly below a living child. In other cases, *if simple traction or manual dilatation fails*, it is preferable to embryotomy if the child is alive. In considering Cæsarian section, it must be remembered that the embryotomy is always a very difficult one, and the death-rate from abdominal section

has been due to sepsis, excepting Andrews' case, where the patient died of exhaustion, and Champneys' case of eclampsia (*vide* table).

In performing Cæsarian section it is often difficult to extract the child. Holland records that Philipps's two cases required trans-uterine embryotomy, and other operators have had to prolong the uterine incision so as to cut through the ring before the child could be delivered (Gilles, Holden, Watts [two cases] Duchamps (19)). Thus all extra-peritoneal Cæsarian sections are contra-indicated, and *in septic cases*, if simple traction or manual dilatation fails, the operations indicated are Cæsarian section followed by hysterectomy if the child is alive, or excision of the gravid uterus unopened if the child is dead.

(6) Cases Treated by Abdominal Section.

Operator.	Parity.	Presentation.	Treatment.	Result: Child	Result: Mother	Notes.
Cameron (30)	?	?	Cæsarian section	L.	L.	Recorded by Jardine.
Champneys (36) *a*	M. 2	?	,, ,,	D.	D.	Death from eclampsia.
,, *b*	P.	Vertex	,, ,,	D.	D.	Death from peritonitis. Both recorded by Routh (36).
Dickinson (18)	M 1	,,	,, ,,	L.	L.	
Gilles (23)	P.	,,	,, ,,	L.	L.	Ring incised during the operation.
Holden (29)	P.	Brow	,, ,,	L.	L.	Ring incised during the operation.
Jardine (30)	M. 4	Vertex	,, ,,	L.	L.	
Philipps (28) *a*	M. 2	Transverse	,, ,,	D.	L.	Transuterine embryotomy required in both cases before the child could be delivered. Both recorded by Routh, also by Holland.
,, *b*	M. 11	,,	,, ,,	D.	L.	
Watt (18) *a*	P.	Vertex	,, ,,	L.	D.	In both cases the patient died of sepsis; in both forceps failed to deliver, and in both the ring required incision and persisted after delivery of the child.
,, *b*	P.	,,	,, ,,	L.	D.	
Boquel (6)	M. 12	Transverse	,, ,, and subtotal hysterectomy	L.	L.	Child died of meningeal hæmorrhage the same day.
Duchamps (19)	P.	Vertex	Cæsarian section and vaginal hysterectomy	L.	L.	Ring required incision.
Gauland (22) *a*	M. 9	Transverse	Embryotomy and hysterectomy	D.	L.	The uterus was ruptured during embryotomy and then treated by hysterectomy.
,, *b*	M. 1	,,	Porro	Macerated	L.	Uterine contents infected.
Rudaux (37)	P.	,,	,,	Macerated	D.	Mother died seventh day of peritonitis.
Sinnetamby (39)	M. 1	,,	Cæsarian section and hysterectomy	L.	D.	Mother died after thirty-four hours.
Andrews (1)	M. ?	Vertex	Partial embryotomy and excision of gravid uterus	Macerated	D.	Death from exhaustion after difficult embryotomy.
White	M. 6	Transverse	Excision of gravid uterus	D.	L.	Specimens Nos. 583 A, B, and C, University College Hospital Medical School Museum, London.

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(47) WILLETT.—'Proc. Roy. Soc. Med.,' 1912, (Obstet. Sec.), p. 335.

(48) WILLIAMSON.—*Ibid.*, p. 337.

(49) ZWEIFEL.—'Therapeutische Monats.,' 1909, p. 129.

April 21st, 1913.

To determine whether Ankylosis of the Jaw is Unilateral.—Kirmisson has often found it difficult to ascertain whether both sides were affected in case of pronounced ankylosis of the jaw. He has obtained useful information from the fact that the lower jaw can be moved sideways, when it is impossible to open the jaws if the lesion is on one side only. If the ankylosis is bilateral, the patient will be unable to move the jaw in any direction. Another sign of unilateral ankylosis is that the cheek on the affected side seems more rounded than on the sound side. This paradoxical phenomenon is due to the fact that the bone support on the side of the ankylosis is more or less atrophied, so that the soft parts developing normally are squeezed into prominence, as it were. On the sound side, the bone being normal, the soft parts stretch normally over them and thus appear sunken in comparison with the bulging soft parts on the side of the ankylosis.—*Journ. A.M.A.*, vol. lx, p. 949.

A LECTURE ON SOME OBSCURE AFFECTIONS OF THE FOOT.*

By A. H. TUBBY, M.S., F.R.C.S.,
Surgeon to Westminster Hospital.

GENTLEMEN,—To-day I wish to lecture to you upon some obscure affections about the foot. The affections of the foot are neither all talipes nor all flat-foot. In these times of stress people's feet are becoming more and more a source of trouble, and I shall therefore speak to you about some of the rarer affections which we meet with.

PAINFUL FEET.

People come complaining of pain in the feet, and it is your business to find out exactly what is the cause. The first group of cases are what may be called those due to *general affections of the health*, or constitutional causes. The most common are associated with gout in elderly people. I find there is, however, a strong tendency amongst practitioners when they do not know what is the matter to tell the patients they are gouty, and give them drugs for that condition and send them away. A painful condition which is often diagnosed as gout is really Morton's disease, anterior metatarsalgia, or metatarsal neuralgia, which affects the front part of the foot, and seldom, if ever, the big toe. Its usual site is about the fourth toe, and the patient is naturally puzzled to know why a pain about the fourth toe and not the great toe is called gout. Nevertheless there is no doubt that people who are gouty have painful feet, because the ligaments and muscles in the feet become relaxed, the arch begins to fall and these patients show the signs of oncoming flat-foot, which are extremely painful. It is a fact that feet which are becoming flat are often much more painful than those which are actually flat. Therefore we must remember that gout may be the sole cause of the pain or there may be a condition of flat-foot following gout. An analogous state is flat-foot following rheumatism, whether it is acute, subacute, or chronic. Rheumatism also gives rise to fibrosities of the plantar fascia, and another condition which causes great pain, especially in young adults, is what is known as gonorrhœal rheumatism, although there is no such thing—it is a gonorrhœal affection of the plantar fascia. Such are the more usual constitutional causes of pain in the soles of the feet.

You meet with pain locally under other conditions, one of which I have already mentioned, namely flat-foot, and the reverse of that state, highly-arched feet. The pain in flat-foot is rather curious and capricious. You will find it, for example, very commonly in the sole of the foot, and it seemed to me that the deeper structures such as the muscles and ligaments have given way, but the fascia refuses for a time to yield, and therefore the main strain in keeping up the arch of the foot is thrown on it, and its various branches and divisions; as these are unyielding and very sensitive acute pain is produced. Other people in whom flat-foot is present have pain on the outer side of the feet in front at the external malleolus, and I have been puzzled as to the cause of it. When the foot is flat it is abducted and everted, therefore the portion of the astragalus which is brought into contact with the external mallelous is one on which there is no cartilage, that is to say it is not a normal portion of the articular surface. We know from experience that portions of bone which are covered by articular cartilage are constantly touching one another, and so long as the cartilage and the bone is healthy we do not feel pain, we know also that if there is slight subluxation it remains very painful for a long time until it is adequately reduced. In my opinion the pain at the outer ankle in flat-foot is due to the fact that two portions of bone which come into contact are not both covered by cartilage. Again, there are all sorts of pain in flat-foot, about the front of the foot as well as in the sole of the foot. An important point about flat-foot is that patients go to bed with their feet aching a good deal; whilst in bed the pain passes away, and when they get out of bed in the morning the pain is most acute. The explanation of that is quite simple and straightforward. When they rest in bed, the component parts of the arch resume their normal position, and the stretched muscles and ligaments recover, and so long as they continue to lie they are comfortable. But when they get out of bed there is a sudden strain thrown on the weakened structures and they begin to stretch and cause acute pain. They tell you that as they go about during the morning the pain becomes better as the parts accommodate themselves. But as the day goes on

* Delivered at the Polyclinic.

the weight of the body begins to have its effect on the structures again, so that the pain again becomes worse.

Quite the reverse condition of flat foot is the foot with a high arch, in which the patient's weight is borne on a small portion, at the heels, on the heads of the first and fifth metatarsal bones and on the outer side of the fifth metatarsal bone—that is to say practically on four bony points, instead of being distributed as it should be. These patients complain of pain, and most of that pain is felt beneath the heads of the metatarsal bones—that is to say, in the ball of the foot—and there is evidence of extreme pressure there, as is seen by the presence of a number of corns.

Some patients will say they have pain localised in the foot, and often in the centre of the arch of the foot. If you pass the finger along the plantar fascia, you come to a spot which is excessively tender. Perhaps you will scarcely feel any thickening, or none, but you will hear from them that they have jumped from a height and felt something give way in the foot, and that something is nearly invariable a few strands of plantar fascia. If the plantar fascia is damaged in this way blood is effused, and this blood becomes organised into fibrous tissue, and caught up in this fibrous tissue are a number of nerve-fibrils, which become pressed upon or stretched and tender. There are other causes which are still more obscure, which I hope, however, to enlighten you upon. They are circulatory causes due to Raynaud's disease, and that curious circulating condition known as erythromelalgia which is an ally of Raynaud's disease. Another condition which is rare and has been recently described is intermittent limping, of which I shall have a good deal to say. So much for pain in the sole of the foot.

Now let me speak more particularly about *painful heels.* A painful heel is a very common affection. The first thing to ascertain about it is whether one or both heels is affected. If one heel it is generally due to some local cause. If both heels are affected it is generally due to some systemic cause. But it is not always so: you must not pin yourselves down to this dictum. The most frequent, I think, of all local causes is that the patient is short in one leg. If you measure the legs of people who complain of pain in one heel you will constantly find a difference of from a quarter to half an inch. And the reason there is pain in the heel is that when they stand they bear most of the weight on the heel of the short leg, and the structures become sensitive in the way I shall tell you of presently. Some patients complain of pain in both heels, and if you make inquiries you find that they are accustomed to stand for many hours. Such people are compositors, letter-sorters and policemen. I have had compositors under care with extremely troublesome heel pain. Then we have cases which are due to such conditions as epiphysitis. One not uncommon cause of painful heel in young people, especially when affecting both sides, is epiphysitis of the os calcis, that is, of the posterior epiphysis of the os calcis which makes the prominence of the heel. This is associated with either rickets—and I have seen young boys with it who were the subjects of rickets—or tubercle, and in some adults it is associated with syphilis. Painful heels are often seen after trauma. For example, a patient jumps from a height and bruises his heel, and the pain persists even after the bruise goes. Pain in the heel is also met with in some forms of flat-foot, and it seems that strain on the plantar fascia is felt, not in the middle of the plantar fascia, but at its attachment to the tuberosities of the os calcis. Then pain may be present without our being able to discover a lesion, as in people who are said to be subject to rheumatism. In such cases the diagnosis is sometimes made by exclusion in the first place, and secondly by noting the effect of iodides, salicylates, etc. Such are some of the most common causes of pain in the heel. One other cause I wish to dwell upon, which we have only verified by radiographic examination. A small bony spur from one of the tuberosities is seen tailing off into the attachment of fascia, as if the posterior attachment of plantar fascia had become ossified. In my experience these cases are very difficult to treat satisfactorily.

When the painful heel is due to local causes at an exploratory operation you will find two or three conditions present. In those people who have stood for a long time and have acquired these painful heels the normal fat is converted into thick dense wash-leather stuff, a greyish mass of fibrous tissue, and there is no doubt that in this thick dense mass of fibrous tissue there are nerve-fibres included, which undergo compression between the bone and the boot; and in operating it is necessary

to remove this thickened subcutaneous tissue completely. Another condition is that in which a false bursa is developed, which becomes inflamed. And the third condition which you find at operation is a spur, and these X-ray pictures show this bony spur. With regard to the treatment, if the case is in your opinion due to constitutional causes, you will use the appropriate remedies. If it is due to epiphysitis, the best thing is local rest, especially in rickety cases. If there is reason to believe that the epiphysitis is tuberculous, then local rest, the application of Scott's dressing and tuberculin should be used. If it is syphilis, local rest and the proper treatment for syphilis. Whatever the cause, local rest plays a considerable share in relieving it. If you find that there is a spur present, my opinion is that it is best not to delay, but to cut down at once upon it and remove the spur. And the line of incision which I find best in these heel cases is a semicircular one, making a flap with its convexity backward. The incision goes straight down to bone around the heel. The flap is then reflected forward, and as the various layers are divided their condition is noted. If you make only a small incision the tissues are too thick and non-retractile to enable you to see properly what you are doing. There is very little hæmorrhage, and the scar, being just above the edge of the foot, is not worried by pressure of the boot. If you find a thickened pad such as I have spoken of, dissect it out: do not take away too much, but just the thick part, and leave as much normal fat as you can, because your patient must stand on the heel after you have operated upon it, and it is not good to have bone left in close contact with skin. Take care to have a good plain surface of bone for the patient to bear his weight upon, and therefore, shape a flat under-surface of the os calcis.

There are some other curious cases of pain in the heel and in the points in the foot. I have had to deal with two or three of these cases, and they are very troublesome. At any rate, I found them troublesome, until by a little ingenuity I found a method of treating them. Three ladies, of neurasthenic tendencies and extremely ill nourished, have been to see me, and the soles of their feet have been a mass of small projecting bones, the tissues wasted away and the fat gone; so that the bony points are so sharp that they almost came through the skin, and prevented the sufferers walking. I was puzzled for a long time to know how to deal with them. I conceived the idea that it would be an excellent plan for them if they were to walk on a water-bath of some sort, and I had a special pair of boots devised, the inner side of the sole being lined with a glycerine bath, which supplies the place of the natural fat, and affords considerable comfort in walking.

It is not always necessary to operate for painful heels, especially if there is no spur present, and you can sometimes overcome the difficulty by various devices. Moreover, it is not always that patients will let you operate. You can have special boots made. First cover the outside of the heel with rubber, then it is useful to have the heel counter-sunk, and this space filled with spongy rubber, so that the patient walks on an elastic bed, and he does not feel the hard contact of the foot with the ground. When you see a case of painful heel you must make up your mind whether you are dealing with a constitutional or local or mixed state. Next, I advise you to have an X-ray photograph taken so as to eliminate this question of spurs of bone; if you do find spurs of bone you will find most palliative treatment is unsuccessful, and the case demands operation, although a few may be treated in the way I have shown you. I advise you to give good trial to palliative treatment if you are sure there is a constitutional condition, especially a gouty or rheumatic condition.

Localised Painful Lipoma.

Now there is a still more rare condition, one which has not been very well recognised, although it has received attention since I first described it. There are two kinds of fatty growth—call them new-growths if you like—which affect the sole of the foot: one is congenital, and I shall call it congenital diffuse lipoma, and the other is localised, and this I shall call localised painful lipoma. Congenital diffuse lipoma is one example of gigantism or overgrowth, and you will find one toe after another becomes enormously enlarged. The greater part of the enlargement is in the subcutaneous tissue, but in the true form of gigantism every tissue of the affected toe becomes enlarged equally. In the congenital diffuse lipoma there is no overgrowth of the bone, but an enormous overgrowth of subcutaneous fatty tissue. It begins at the

extremities of the toes and proceeds steadily upwards along the limb. This is a most striking point to me, that although microscopical examination shows that it has no malignant appearances, yet clinically it behaves exactly like one. If you dissect it out, and take away every particle of fat which you can see, the growth recurs. If you amputate the digit—the toe or the finger, for it affects the fingers as well—you will find that it reappears further up the limb. If you allow it to grow unchecked its growth is rather curious. It proceeds for a certain time in one continuous line, and some distance away up the limb there appear three or four fatty nodules, which coalesce and join with the original growth. So it gradually goes on, and unless you amputate the part a considerable distance up the limb, you will find the fatty growth recurring in the scar, and even higher up. And therefore I am right in saying that whilst it has none of the microscopical characters of malignant disease, clinically its behaviour is exactly that of malignant disease, because it progresses and renders the limb useless. As far as I know there are no metastases. This diffuse lipoma is a very interesting condition because of the deformity to which it gives rise.

I now pass on to speak of localised painful lipoma. Diffuse lipoma is not painful but the localised form is, and I have seen five. It begins almost invariably just below the internal malleolus, actually in the arch of the foot. Frequently it is in people who stand a great deal, and I have seen one case very well marked in a nurse. The pain to which it gives rise is great, and a striking fact is that often these abnormal fatty growths are associated with extreme pain. These patients have been sent to me for flat-foot, the reason being that the arch of the foot is practically obliterated by the fatty growth, and it looks as if the arch had fallen. So that you must be on your guard with these cases and not mistake them for flat-foot. The only treatment is to dissect the lipoma out with care. The first point to remember is that although they are fatty they are extremely vascular and will bleed most freely. And the second point is that the lobes of fat of which they are composed are different from the surrounding lobes: the fat here is more yellow, and the lobes are three or four times the size of those in the normal foot. When you come to examine them microscopically you find there are other features. The connective-tissue cells between the fatty lobules are increased both in number and size, but still more important it is to notice that there is a very wide ramification of thick-walled vessels; the walls of the vessels are thicker than usual, and much more abundant and more widely spread. Hitherto I have never seen the slightest trace of anything to make me suspect I was dealing with anything malignant. The point is that whilst this growth remains localised, and grows larger, it never throws out islands and processes as the congenital diffuse lipoma does. Then, harking back to the fact that abnormal fatty growths are very painful, I think the pain in this case is due to the presence of nervi vasorum in abnormal number. I have never been able to demonstrate any nerve-endings in the fat itself. If you remove these localised painful lipomata they do not recur. Therefore they present a contrast with the congenital variety. I had to deal on one occasion with a very curious nævo-lipomatous condition, which occurred in the hands of a fairly young woman. It began as far as I remember in the second metacarpal space of the left hand. The part looked blue at first, was swollen, and gave the impression on palpation as if it were composed partly of fat and partly of blood-vessels. The most striking fact was that the tenderness was extreme, and she could not bear anyone to touch it at all. As time went on the nævoid element became more prominent, the part was reddish-blue, and evidently it was very fully supplied with blood-vessels. I removed it on the first occasion, but it recurred. A colleague removed it in my absence on a second occasion; again it recurred. Then the same condition commenced in the other hand, and there were several recurrences, and so we gave up the idea of removal, and tried, I think, static currents. Since then I have lost sight of the girl. Here, then, was a case in point of fatty nævoid growth which was very tender. I think that case throws light on these example of localised painful lipomata, and they show you that when there is abnormal development of fat and nævoid condition of vessels there is extreme pain and tenderness also.

Malignant Growths.

I need only speak very shortly of malignant growths of the feet. Everyone knows that corns

and warts, if irritated, are particularly liable to degenerate and form epitheliomatous growths. But from time to time one meets with a case of sarcoma of the foot, and I remember such a case some years ago. It was a lady who had a swelling occupying the arch of the foot, and I thought from the history that it was periostitic. I cut down upon it, and to my horror I found it was a spindle-celled sarcoma. She was seventy years of age, and there was nothing else but to obtain permission to amputate the foot on a subsequent occasion. So I warn you to be on the look-out for sarcomatous growths and gummata in the feet.

Achillodynia.

Now, when I mentioned to you pain in the heel, I meant pain on the under surface of the heel. I will now speak of conditions of pain in the back of the heel, the so-called achillodynia, which is not uncommon, and it may be due to local causes or to general causes. I will first of all speak about the anatomy. Superficial to the tendo Achillis there is a bursa, and between it and the upper part and the posterior surface of the os calcis there is another bursa. Either of these may become inflamed, and when the anterior bursa is inflamed it is anterior achillodynia, and when the posterior is inflamed it is called posterior achillodynia. When the anterior bursa is inflamed you can feel a projection on either side of the tendo Achillis and there is a fluctuation across beneath the tendon. If it is in the posterior bursa there is superficial fluctuation. The causes of the bursitis may be either general or local. For the general causes we go back to gout and rheumatism, and for local causes in children we think of overgrowth or too rapid growth about the epiphysis—a sort of physiological epiphysitis. And you may have inflammation of the anterior bursa associated with tubercle, and later there may be inflammation, associated with syphilis. The local causes are traumatic, such as a kick on the back of the heel through football, or the heel is struck in a game of hockey. Occasionally strain of the tendo Achillis causes bursitis. The treatment is comparatively simple and straightforward: rest, counter-irritants, absorbents such as Scott's ointment, and patience. If the condition does not improve, and if you have tried everything you can—and whilst you are waiting, of course, you will have an X-ray picture taken—and if there is evidence of tubercle or osteitis, you will be justified in cutting down on the bone and gouging out the affected part and removing this bursa. In several cases of tuberculous affections of that sort I have gouged out this epiphysis so far as is necessary to remove these bursa. Some of these cases are so troublesome and persistent that they come to operation, and the operation for the condition must be done on these lines.

Fracture of Base of Fifth Metatarsal Bone.

Some people suffer from a curious condition of pain about the base of the fifth metatarsal bone, and it is well recognised and due to definite causes. It may be sudden, following on a strain on the foot, a sudden movement of the foot, particularly if you take a leap off one foot. Or a man who is getting on into middle age may suddenly wish to show that he can still play cricket and plays at point, and wishing to show his activity leaps about. In two or three instances such people have been seized with pain about the base of the fifth metatarsal bone. For a long time the cause of this was unknown, and it was only revealed by X rays. When a man has his foot in the position I show you, with the great toe turned somewhat inwards, he throws a "cross-breaking strain" on the fifth metatarsal bone, and the bone fractures across at the proximal part. It only requires rest and time and it will heal. Many patients have that fracture without known cause. They suddenly feel acute pain there, and the bone snaps. It is usually due to a sudden definite movement carried out with the foot inverted, when most of the pressure comes on the fifth metatarsal bone. That is not an uncommon form of fracture.

Another cause of pain about the fifth metatarsal bone you find occasionally in Morton's disease, that is to say, metatarsalgia. The foot acquires a curious twist inwards, and the base of the fifth metatarsal bone is unduly prominent and is subject to pressure by the boot.

Circulatory Affections of the Feet.

I want to speak now of circulatory disturbances of the feet which cause persistent and troublesome pain. And the most usual example of circulatory disturbance is arterio-sclerosis in persons of advancing years. Elderly gentlemen occasionally consult me because of persistent pain situated in all

parts of the foot, and when I know that the pain is in every part my suspicion is aroused, and I take care to feel the vessel, to ascertain whether there is any pulsation in the posterior and anterior tibial arteries. Often I find that the pulsation in one or other of them is absent. This condition is often the forerunner of senile gangrene, and if the patient lives long enough he may suffer with that—that is to say, the arteries are becoming more and more sclerosed and closing up. This oncoming gangrene is often associated with extreme pain, and I strongly suspect it is due to the development of certain chemical substances which irritate the nerve-endings. You must be prepared to do something, and what I have found most valuable in the treatment is to keep them warm and give $\frac{1}{8}$ gr. of codei, which I learnt from the late Dr. Pavy when I was his house-physician. This should be given two or three times a day. After giving it the pain disappears, the feet become warmer, and the circulation seems undoubtedly to improve. You may in this way tide people over their trouble, and I think it has some influence in averting an early attack of senile gangrene.

There are two other conditions I should like to say a word about, namely, Raynaud's disease, which of course affects much younger people, and is generally accepted as due to a spasm of the muscular coats of the arteries, and that curious first cousin of Raynaud's disease known as erythromelalgia, in which you find the pain and the flushed condition of the foot. And the flushing is in patches, and these patches afterwards run together, and you will have a flushed, painful, sensitive sort of foot. The foot is not wet with perspiration, but is usually dry. These cases are most difficult to treat, and the only thing I could advise a lady who had it was to have galvanism and faradism constantly applied. Massage also seems to have some influence, but drugs appear to have no effect. They are troublesome cases to have to deal with.

The last affection I intend to speak about to-day is a rare one, known as dysbasia angio-sclerotica, or intermittent limping. This is an affection, not of advanced years, but of young, middle life, and it begins with pains in the calves, extending down to the feet. And this pain is much worse when the feet are allowed to hang down; and you will find the feet are pale and often cold to the touch; they are also dry and not sweating. This condition goes on, the feet becoming colder and the circulation gradually ceases, and the end of it is gangrene. It occurs more commonly in males than in females, twice as often, and between the ages of seventeen and forty years. A striking fact is that it seems to affect Jews more than it does Gentiles. There is one condition which is closely associated with it, viz. the abuse of tobacco, and it is found that 60 to 70 per cent. of those who are affected with it are ardent smokers of tobacco. The earlier cases have improved when they have given up the tobacco habit. This is, then, a very curious condition, beginning with pain and cramps in the calf, which are probably expressive of change in the posterior tibial artery and tender calf, and the occlusion of the circulation, ending in gangrene. The local conditions of arteries found are of two kinds: either the middle coat of the artery is converted into calcareous masses so that the intima gives way and the channel is blocked by a mixture of calcareous masses and broken-down intima, or there is the curious condition of proliferative endarteritis in the middle coat. The connective-tissue cells and the middle coat develop enormously and gradually replace the muscular coat, and, I suppose, ultimately cause it to disappear. Finally the channel or lumen of the artery is blocked up by this proliferative endarteritis. I had a striking case which came under my notice at Westminster Hospital eight years ago: I have not yet published it. It was the case of a Jew, æt. 22 years. He first came to my out-patients, with pain in the calves. Knowing the history of this affection, I watched him and we tried all kinds of treatment. The pain in the calves disappeared for a time. But later he turned up again with intense pain in the feet, which were cold, and the pulsation of both tibials was lost. The next stage was gangrene of the toe. We kept him in and watched him. The pain increased and the gangrene spread rapidly in one leg, and I advised him that the only thing was amputation. I amputated through the popliteal space; and when we came to examine the arteries we found this marvellous proliferation of the middle coat, and the arteries from the bifurcation of the popliteal downwards were absolutely blocked: there was no circulation at all. Yet this was not the worst. He had a year or two freedom, and then the other foot began to go like the first one, and I had to amputate through the popliteal space in that one, and then his trouble ceased. I quote this case to you to show that it is a terrible disorder. As far as I remember, this unfortunate Jew did not smoke much.

I have chosen for the lecture to-day a series of subjects which I believe you will find useful in general practice.

April 21st, 1913.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from page* 32.)

(194)

Relieve Pain where possible.

There is no doubt about this, that you should if possible relieve the patient's pain. I remember I was very much impressed by an old doctor, who practised for many years, telling me one day, "I never let my patients suffer pain." That is a very good rule to follow. There seems to be such a fear of prescribing opium or morphia nowadays because of the danger to the patient of becoming a morpho-maniac. But it is as inconsistent to believe that a patient to whom you give morphia will become a morpho-maniac as it is to believe that every woman who has a glass of beer will become a drunkard.

(*Ibid.*)

[N.B.—This is a rule which must be followed with the greatest caution in the acute abdominal case.—F. W. L.]

(195)

Treat the Patient behind the Disease.

In conclusion, I want you to take away at least one thing from this lecture, and that is, the great value of general treatment in gynæcological cases. Do not go on playing about, painting the uterus, whilst forgetting the woman who possesses it.

(*Ibid.*)

(196)

Typhoid,
or
The "Outward and Visible Sign."

It is one of the most remarkable things in medicine that if you see a rose spot on the abdomen, which any untrained observer would pay no attention to, you can say there will be ulcers in the intestine. And if outsiders are inclined to jeer at medicine, ask them if they can show anything to compare with that. It is one of the triumphs of observation in medicine that the presence of these little round spots on the abdomen should indicate that there is ulceration in the intestine.

("Typhoid Fever," by W. Hale White, 'Clin. Journ.,' vol. xxxvii, p. 129.)

(197)

Abnormalities not necessarily Pathological.

Such individuals may be normal in every other way—physically, mentally and sexually; they are mannikins or diminutive men and women, or better, so it seems to me, they are dwarfs or dwarfish people; there is no evidence that they are pathological results.

("Ateleiosis (Hastings Gilford's form of Essential Infantilism,)" by H. Batty Shaw, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvii, p. 136.)

(198)

The Wassermann Reaction and Ante-nuptial Syphilis,
or
"Let the Dead Past bury its Dead."

The test for syphilis, as most people know, is the "Wassermann reaction." This may be very accurate, but it is not one which the general practitioner can himself use in his daily work. The history must be necessarily incomplete, because you cannot run the risk of sowing discord between husband and wife by suggestive questions as to the ante-nuptial history of the other partner. So if you get a married woman who has had two or three abortions, it is best to assume the previous occurrence of syphilis and not worry the patient about the history.

("Abortion," by G. E. Herman, M.B., F.R.C.P., F.R.C.S., 'Clin. Journ.,' vol. xxxvi, p. 145.)

(199)

Clinical v. *Laboratory Diagnosis.*

One is, however, I think, entitled to regret that the assistance of the clinical expert is now so often replaced by some form of tuberculin test, or by examining the sputum for bacilli which are not likely to be present until softening has commenced.

("Some Points in relation to Tuberculosis and its Prevention," by J. Edward Squire, C.B., M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvii, p. 161.)

(200)

An Englishman's House is his Castle—unless he happens to be poor.

The poor are sadly over-visited and get accustomed to the intrusion of uninvited strangers, but they often resent the visit of officials, who may claim admission as a right. For this reason there may be an advantage in making use of voluntary agencies to supplement the official sanitary inspectors or health visitors (in cases of tuberculosis).

(*Ibid.*)

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THE VACCINE TREATMENT OF SEPTICÆMIA.

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WHEN vaccine therapy was first introduced its applicability was thought to be limited to the chronic localised infections, for it was not unnatural to suppose that in general systemic infections the mechanism of immunity would already be overtaxed, and to introduce a further supply of bacterial poison would be to add fuel to the fire, and to incur a responsibility too great to be contemplated. With more experience it soon became apparent that this *à priori* reasoning did not fit the facts, and that the entrance of bacteria into the blood-stream, so far from exhausting the mechanism of immunity, apparently had little power to stimulate it at all, and that the protective substances found in the circulating blood had their chief seat of origin elsewhere. A vaccine introduced into the subcutaneous tissue would then not necessarily do harm, and might do good by causing protective substances to be formed which would be carried into the blood-stream. This hypothesis has served a useful purpose in making us less afraid of the use of vaccines in acute diseases, but while it seems to offer a rational explanation of the beneficial results of vaccines in those cases of septicæmia in which no local infective focus can be found, it is difficult to accept it when the blood condition is secondary to a wide-spread local infection such as cellulitis, uterine infections and pneumonia, and yet vaccines are equally of service in such cases. It must be recognised that we have no satisfactory explanation of the action of vaccines, and their use is at present empirical.

With an acute case it is always difficult to connect the result, whether successful or unsuccessful, with the treatment, but although cases

of septicæmia may get well without any treatment, this is a result we do not usually expect, and the condition is rightly dreaded as one of the most dangerous we are claled upon to meet. Those who have used vaccines in the treatment of such cases can relate many instances of striking success in which improvement was so rapid and startling, as shown not only by the temperature chart, but by all the clinical indications, that it is difficult to attribute it to any other cause than the vaccine. Certainly after seeing many such cases I approach the treatment of an acute septic case with a confidence which was before quite lacking, and I believe that in the near future the systemic infections will prove the most successful field for the skilled employment of vaccines.

For the purposes of this paper I have selected certain cases which are either of special interest or illustrate the type of case in which vaccine treatment may meet with success. No attempt is made to describe all the septicæmic cases treated.

In all the cases described bacteria were found in the blood by cultures made according to the usual methods. Cases which clinically would be described as septicæmia, but in which no blood-cultures were made, or if made, proved sterile, are excluded. Possibly some of the cases may not be regarded as true septicæmia, if this term be limited to the cases in which bacteria are not only present but multiplying in the blood-stream, but in all the cases a growth was obtained from 5 to 10 c.c. of blood withdrawn from a vein, and added to broth or agar, and I know no method by which the fact of multiplication of bacteria in the blood can be differentiated from their mere presence without multiplication.

An attempt was always made to isolate the micro-organism from the primary focus of infection when this could be determined, and in most cases a bacteriological examination of the urine was made, as not infrequently the infecting agent can be isolated from the urine when blood-cultures are sterile. The micro-organism found in the blood was in most cases used for the preparation of the vaccine, as it is reasonable to suppose that it would be more virulent than one from the local site, and in all the cases the micro-organism was found in the blood in pure culture. If, however, a growth from the local site could be obtained earlier, this would be used for, at any rate, the first inoculation. Vaccines were sterilised by heating at 60° C. for half to three quarters of an hour, and were diluted with 0·5 per cent. carbolic acid in normal saline. No attempt was made to test the sterility of the vaccine before use. In these acute cases every hour is of importance, and one cannot wait the necessary time. The point seems to me rather of academic than practical importance. After thousands of inoculations of vaccines of many kinds I have seen nothing to lead me to think that the vaccines were not sterile. In any case incubation for twenty-four hours is insufficient to test sterility, for the vitality of the germs is so lowered that growth may not occur in this time, though it may after three days' incubation, but such a delay might be fatal in an acute case. It is frequently possible to give the first dose of vaccine eighteen hours or less after the material was taken.

Case 1: *Typhoid fever with* B. coli *septicæmia.*—The first case of septicæmia was an unusual one. On April 27th, 1907, I saw with Dr. Freer, of Old Hill, a gentleman, æt. 37 years, suffering from typhoid fever. The Widal reaction had been positive earlier in the illness. After a rather irregular course for five weeks, during which thrombosis occurred in the veins of the right arm, a severe rigor took place, the temperature rising to 106° F., and the patient's condition became very grave. When I saw him next day he was in a state of deep stupor and prostration, the abdomen was distended, and there was much meteorism, the tongue was dry and cracked, urine was passed involuntarily, and there was slight cyanosis. His condition seemed to us hopeless. Blood was taken and showed the presence of a bacillus, at first assumed to be the typhoid bacillus, but on investigation later it gave the reactions of *B. coli.* A vaccine was made, and on the 29th* a dose of 120 million bacilli was injected. The temperature fell in twelve hours to 99° F., but rose again the following day to 101·6°. On May 3rd the temperature was 102·8°, and a second dose of 120 millions was given. Next day the temperature dropped to normal and remained so, the patient making a rapid and uneventful recovery. The diagnosis of typhoid fever was confirmed, as on May 1st the patient's wife felt ill and later developed a typical attack of the disease.

* On chart inoculation is wrongly indicated as having been given on the 27th.

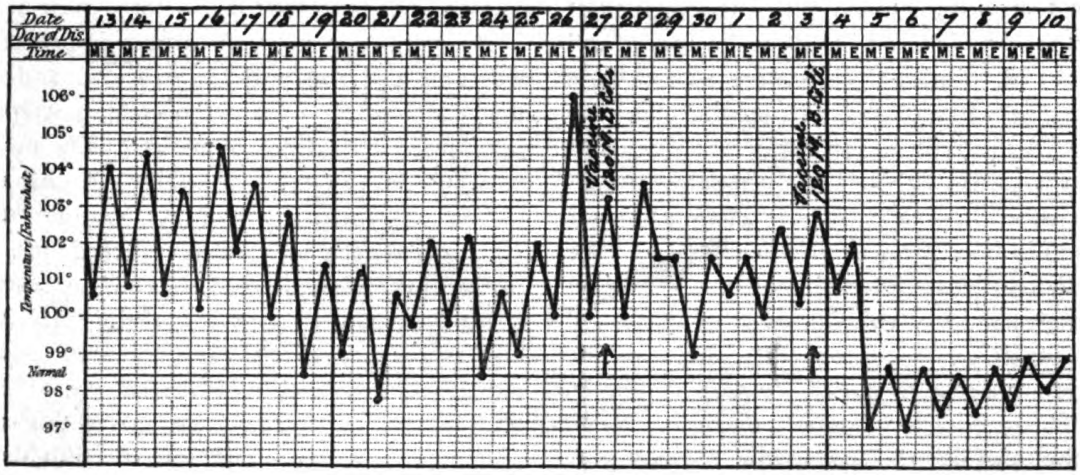


CHART 1.—Case 1.*

This case illustrates several points of interest. Rigors are not common in typhoid fever, and are of serious omen. I had previously suspected that rigors were caused by a secondary infection, possibly *Bacillus coli*, but in an earlier and fatal case no growth was obtained from the blood. A difficulty which at once presented itself in the case was the amount of vaccine to be given. No similar case had been recorded. If a small ineffective dose had been given there would probably have been no opportunity of giving another. The dose actually given—120 millions—is much larger than I should give in a similar case at present, but it is possible that we err on the side of timidity in these acute cases. A large dose is more likely to cut the disease short, whereas small doses must be repeated them several times to obtain an effect. The same difficulty is seen in the treatment of typhoid fever, for the doses recommended by various authorities vary from 15 to 250 millions, and equally successful results seem to be obtained. The effect of a large dose in aborting the disease seems to be shown in the following case, which may be quoted here, although no blood-culture was made.

CASE 2: *Puerperal infection with streptococci and* B. coli—A lady under the care of Dr. Crowe, of Worcester, on the eighth day after confinement had

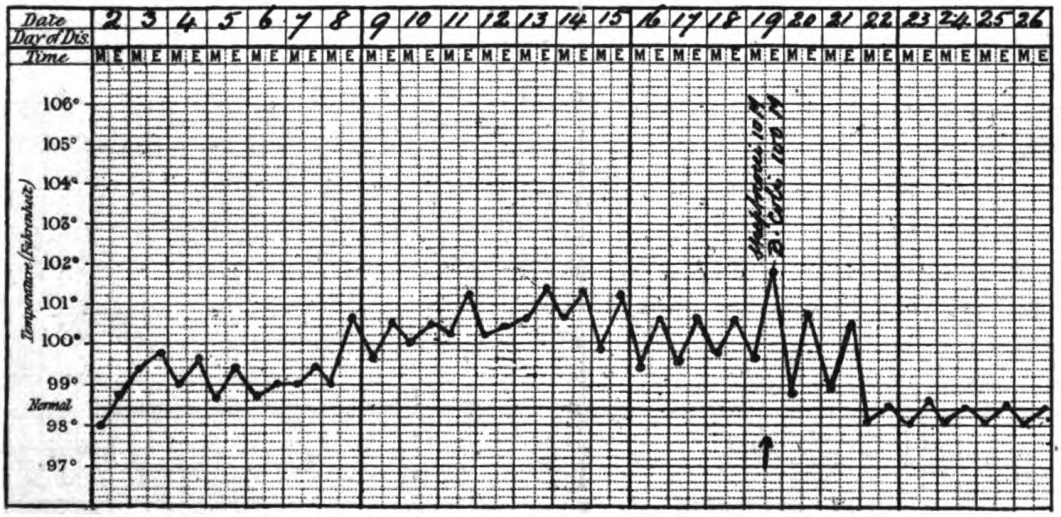


CHART 2.—Case 2.

* We are indebted for these charts to the 'Journal of Vaccine Therapy.'

a rise of temperature to 99·5° F. It remained at this level for five days, and then rose to 100·4°, and later to 101·2°. Infected clots were passed from the uterus, which was washed out with biniodide solution. Anti-streptococcic serum was given five times in seven days without improvement following. Vaccines were then prepared from a streptococcus and *B. coli* isolated from a uterine clot. On the eighteenth day of the fever, the temperature being then 100·4°, a dose of 10 millions streptococci and 100 millions *B. coli* was given. The same evening the temperature rose to 101·8°, afterwards falling to 98·6°. The reaction was followed by improvement, and on the third day the temperature fell to normal and the patient completely recovered.

Four other cases of puerperal septicæmia may be related.

CASE 3: *Puerperal septicæmia.*—The patient, æt. 25 years, was confined for the first time on May 3rd, 1912. The presentation was a breach and the labour a difficult one. There was a large tear in the first part of the vagina. During the next eleven days the temperature was irregular, rising to 100° F. each day, but the patient's condition did not cause much alarm. On May 15th a severe rigor occurred and the patient became collapsed. On the 16th I saw her in consultation. Blood-cultures gave a pure growth of a streptococcus, and from the uterus a streptococcus and *B. coli* were obtained. On May 19th, 21st, 23rd and 25th, 10 million *B. coli* and 5 million streptococci were given. After the second dose the temperature, previously remittent, became intermittent and continued so. With the change in temperature the patient's aspect improved. On May 29th and 30th and June 1st and 2nd a dose of 15 million *B. coli* and 10 million streptococci was given, the doses were gradually increased, reaching on the 8th 30 million *B. coli* and 20 million streptococci. The temperature gradually fell, and after the 11th did not exceed 99°, except on the 16th, when it rose to 102° in association with some vomiting and abdominal pain. A pelvic abscess was feared, but the symptoms cleared up and the patient made a good recovery.

CASE 4: *Puerperal septicæmia.*—In this case the temperature rose to 103° F. on the fifth day after confinement. During the six following days anti-streptococcic serum was given eleven times in doses of 25 c.c. and also two doses of anti-colon serum. The temperature came down slowly, but rose again on the eighth day of the fever. A vaccine of *B. coli* and streptococci had previously been prepared from uterine swabs, and a streptococcus found in the blood. On July 12th 5 million streptococci and 15 million *B. coli* were given, and this dose was repeated on the 14th and 16th. On the 17th there was a decided drop in the temperature, but both hips became painful and swollen, and the next day the articulations of the jaw were painful, and also the back of the neck. On the 20th 25 million *B. coli* and 10 million streptococci were given. On the 21st there were signs of pneumonia, and on the 22nd pericarditis, and the patient seemed *in extremis*. On the 22nd 15 million streptococci and 30 million *B. coli* were

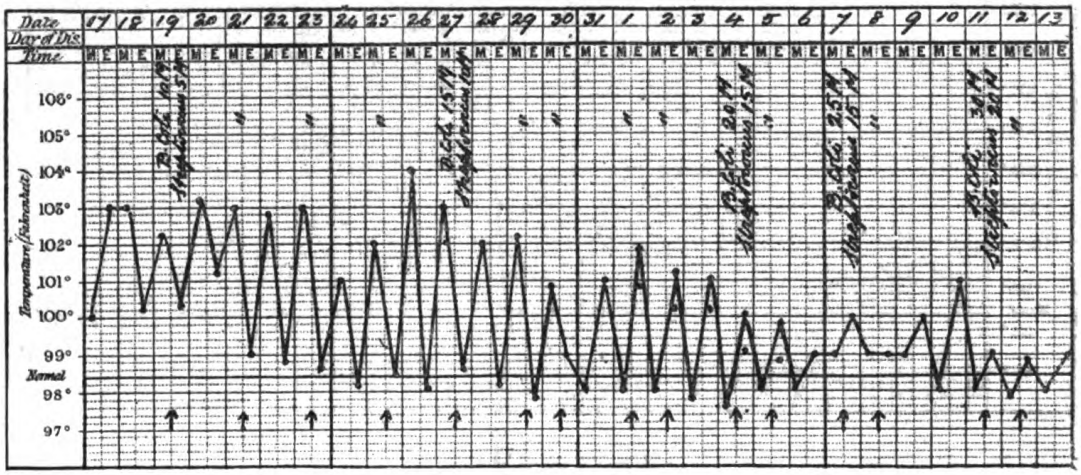


CHART 3.—Case 3.

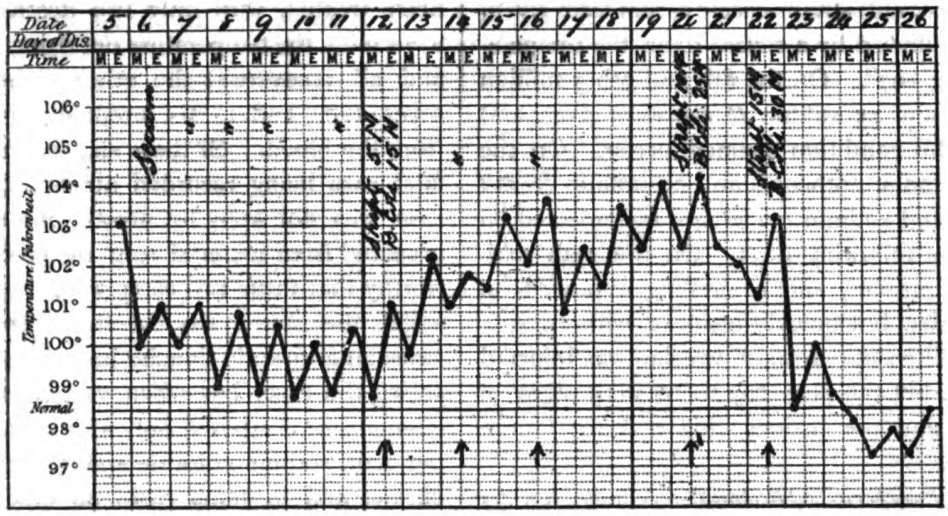


CHART 4.—Case 4.

given, and in a few hours the temperature dropped by crisis. No further vaccine was given, the swellings over the hips and jaw disappeared without suppurating, and the patient rapidly recovered.

CASE 5: *Puerperal septicæmia.*—Patient, æt. 32 years, was delivered of her first child on November 1st, 1911, after twelve hours' labour and with instrumental aid. There was considerable hæmorrhage, and as the placenta seemed delayed it was removed by hand. Eighteen hours afterwards the patient shivered, and the temperature rose to 104° F. A lysol douche was administered, followed by abundant saline. On the 4th the douche was repeated and 50 c.c. anti-streptococcic serum was given. On the 5th there was a severe rigor and the lochia became offensive. The uterus was then curetted and swabbed with pure lysol. On the same day I saw the patient in consultation. The condition was very serious, and patient highly nervous and despondent. Cultures were taken of the blood and from the uterus, and as there was also considerable pyorrhœa, cultures were taken from the gums. Five million streptococci and 15 million *B. coli* were given from vaccines made from a previous puerperal case. The blood gave a pure growth of *Streptococcus pyogenes*, and the same organism together with *B. coli* grew in cultures from the uterus. The next morning the patient seemed better, but in the evening a severe rigor occurred and she became very weak and collapsed. On

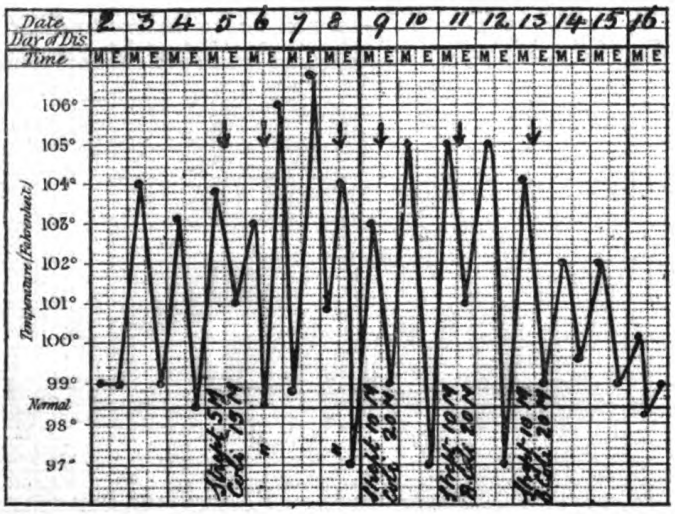


CHART 5. – Case 5.

the 7th 20 c.c. anti-streptococcic serum were given, only to be followed by a severe rigor, the temperature reaching 107°. On the 8th and 9th 5 million streptococci and 15 million *B. coli* were given, and on the 9th the condition seemed better. On the 11th 10 million streptococci and 20 million *B. coli* were given, and sodium nucleinate was injected on this and the following days. On the 13th pericarditis was present, and a swelling appeared on the right wrist, but quickly resolved without suppuration. The same dose of vaccine was injected on the 13th, but the next day there were signs of pneumonia, and the patient died of heart failure on the 16th.

Case 6: *Puerperal septicæmia.*—A lady, æt. 33 years, was confined on July 16th, 1912. On the 19th the temperature rose to 100·2° F., and on the 21st to 103°. The patient was seen with her doctor late that night, and 10 million streptococci and 15 million *B. coli* from a stock vaccine were given at once. A streptococcus was obtained in pure culture from the blood, and together with *B. coli* was found in the uterine discharge. On the 23rd 10 million streptococci and 15 million *B. coli* were given from vaccines prepared from the patient's own germs. The temperature became intermittent, and on the 24th fell to 101°. On the 25th 15 million streptococci and 25 million *B. coli* were given, and on the 27th and 29th 20 million streptococci and 30 million *B. coli*. This case was treated promptly, and in spite of the obvious severity of the condition there was little anxiety as to the result.

In all, thirteen cases of puerperal infection have been treated, with only two deaths. Five cases were mild, the temperature not rising above 102° F., and all recovered; the other eight were of the severe type, and in six streptococci were isolated from the blood. Of these six, five recovered, the fatal case being recorded above; the other fatal case was not seen by myself, the vaccine being prepared from material sent by post. It is noteworthy that these two cases were the only ones in which the uterus was curetted. In most of the cases an antiseptic douche had been given before seeing the case, but when vaccine treatment was commenced I advised only a douche containing 3 per cent. sodium chloride and 1 per cent. sodium citrate, and in some cases no other douche was given. In two cases the streptococcus was of the *pyogenes* type, and one was fatal; in three cases it was of the *salivarius* type, and in all three an alveolar abscess was present from which a similar streptococcus was obtained; in the remaining eight the streptococcus resembled the *fæcalis* type, but did not always ferment mannite. In the two with *Streptococcus pyogenes* it seemed likely that infection was introduced by manipulations; in those with *streptococcus salivarius* and *fæcalis* it seemed possible that the infection might be from the patient herself.

The cases recorded will show the importance of beginning vaccine treatment as soon as possible after infection declares itself. An injection of stock vaccine should be given at once; it can do no harm, and may contain a streptococcus of the same type as that causing the infection. Although serum

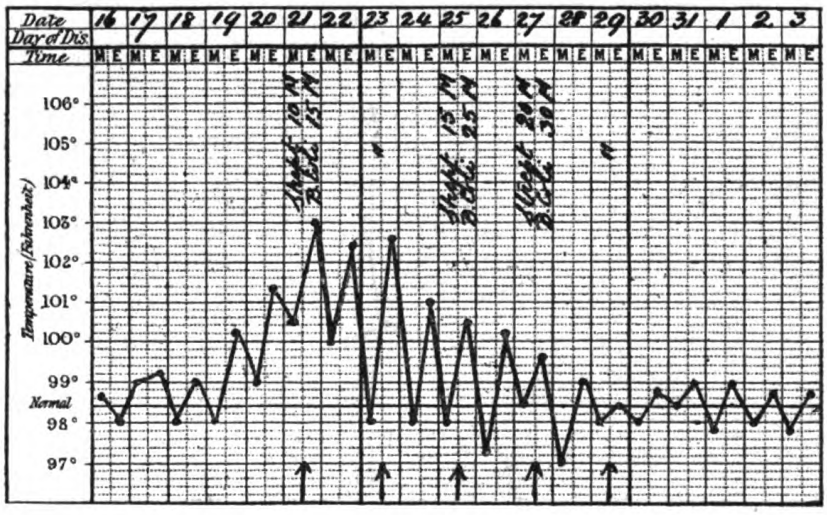


Chart 6.—Case 6.

seems very variable in its action, it also should be given in doses of 50 c.c. as soon as possible until the autogenous vaccine is prepared. If no result follows the first dose of vaccine the dose should be steadily increased. In all septicæmic cases I believe it to be of the greatest importance that inoculation should be made only when the temperature is falling. Theoretically this would seem to be the best time, and it is confirmed by practice. The temperature should be taken at least four-hourly, so that the appropriate time for injection may readily be seen. As this may occur in the early morning, or at a time when a doctor cannot visit, careful instructions should be given to the nurse and the vaccine left in her charge.

Case 7: B. coli *septicæmia*.—On June 25th, 1911, I saw a lady, æt. 44 years, with Dr. Bunting, of Edgbaston. In May she went a sea voyage to Morocco. On May 29th, after leaving Lisbon, 120 persons on board the steamer suffered from severe diarrhœa and vomiting lasting a few days. No food was eaten at Lisbon, and none was taken on board. For two days the patient had much abdominal pain and vomiting, with great muscular weakness, prostration, and aching head and limbs. Her temperature was then 103° to 104° F. After two days intense diarrhœa began with six to twelve movements a day. She was brought home on June 12th. Temperature remained irregular; there was much abdominal distension, but the diarrhœa and pain were less. She was in a dazed, apathetic and depressed condition. Aperients caused several motions, but next day the temperature would be more raised. The tongue, at first clean, was now furred. The abdomen was not tender, the spleen could be felt, and there was a soft mitral murmur. There were several septic stumps in the mouth and one gold-crowned tooth. The blood did not agglutinate the typhoid or paratyphoid bacilli. The leucocyte count was 4800. A blood culture gave a pure growth of *B. coli*, and the urine was turbid, contained a few pus-cells, and many *B. coli*. Vaccine treatment was begun with doses of $7\frac{1}{2}$ millions, increased later to 10, 15, 25 and 40 millions. Patient began to improve as soon as the treatment commenced, and the urine gradually cleared up. An interesting symptom in this case was the severe aching and tenderness of the limbs. The experienced sister in charge remarked that the patient was as tender as a bad typhoid case. This, together with the great muscular weakness, I have noticed in other cases of *B. coli* infection.

Case 8: *Septicæmia*; *pyæmic pneumonia*.—A girl, æt. 17 years, was admitted into the General Hospital under the care of Mr. Haslam, with a painful swelling in the right lumbar region of about a year's duration. Hydronephrosis was diagnosed, and on January 30th, 1908, an incision was made into the right kidney. The kidney contained much foul urine and afterwards became infected. This necessitated a second operation on Februrary 19th for its removal. A few days later signs of pneumonia appeared. Dr. Stacey Wilson was asked to see her, and found consolidation of the whole of the left lung and base of the right. On the 28th I punctured the right lung, and from the small amount of blood-stained fluid obtained grew a streptococcus in pure culture. The blood contained the same organism. On February 28th and 29th the temperature was 104° F. On March 1st 10 million streptococci were injected. The temperature fell to 100·5°, but rose again to 102·5°, and

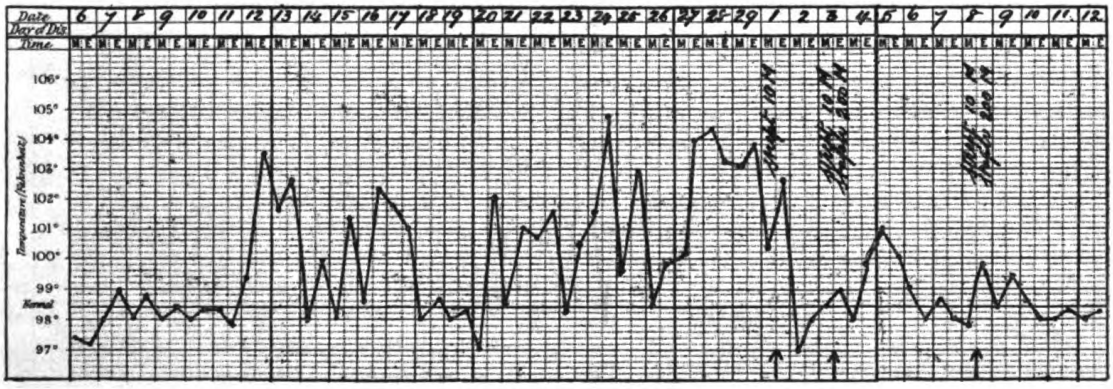


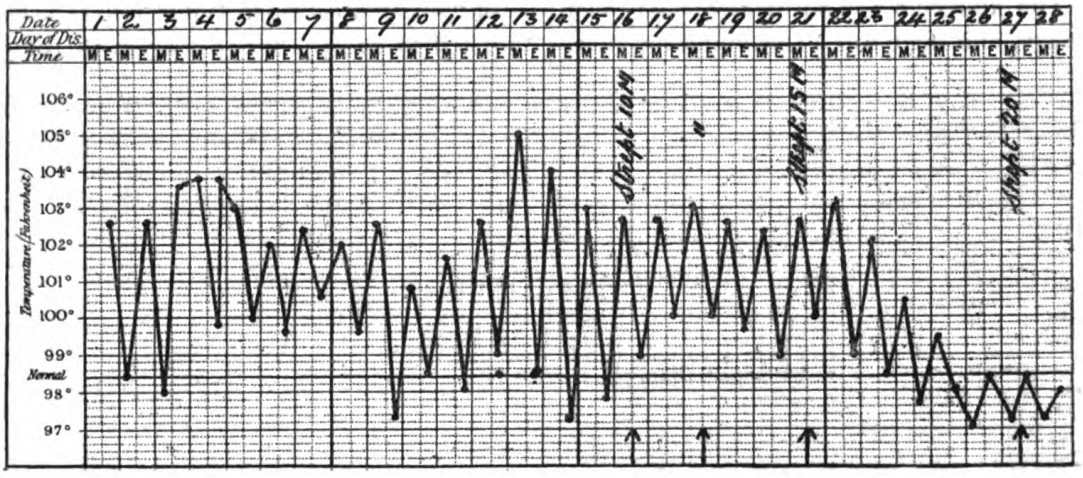
Chart 7.—Case 8.

fell by crisis the next day to 97°. On March 3rd, 10 million streptococci with 200 million staphylococci obtained from the operation wound were injected. There was a reaction to 101°, and the dose was repeated on the 8th with a smaller reaction to 99·9°. The lung cleared in a remarkably quick manner, and the patient made an excellent recovery.

CASE 9: *Cellulitis; septicæmia.*—A medical man infected his finger at a post-mortem on a case of infective endocarditis. In a few hours he felt ill and shivered. The finger became swollen, and the infection spread to the tendon sheath and axillary glands. He was admitted to the general hospital under Mr. Gilbert Barling, who promptly incised the finger. The inflammation rapidly spread along the subcutaneous tissue of the arm, and a severe erysipelatous inflammation spread all over the trunk and down both thighs. The patient became dangerously ill and delirious. From the blood and from the arm a *Streptococcus longus* was obtained. The first dose of vaccine—10 millions—was given on the sixteenth day of the infection, the patient then being at his worst; the dose was repeated on the 18th, 15 millions on the 21st, and 20 millions on the 27th. The temperature reached normal on the 24th. There was improvement after the first injection, and it was interesting to notice after each injection that the margin of the erysipelas on the trunk receded and advanced again as the effect of the vaccine passed off. I think there can be little doubt that the successful result was due to the vaccine treatment.

This patient was nursed in a side ward, and a nurse in the adjoining ward, but not in attendance upon the patient, became infected with severe facial erysipelas, as also did a male patient. Both were treated with the vaccine from this case; the nurse's temperature fell by crisis after the fifth dose of vaccine.

CASE 10: *Resection of nasal septum; septicæmia.*—On March 16th the nasal septum of a young man was removed. The next day the temperature rose to 100·6° F., and remained between 100° and 101° for several days. On the 23rd it reached normal and the next day he was allowed out of bed, but on the 25th there was a severe rigor and the temperature rose to 103°. On the 26th he complained of a painful swelling over the right lower ribs and of pain and swelling at the back of the neck. On the 27th these had increased, and the next day an incision was made into the swelling on the right side, but no pus was found. This was followed by a rigor and a temperature of 105°. I saw the patient for the first time on that day, and made blood-cultures from which a streptococcus was obtained. A vaccine of 5 millions was given on the 29th and 30th, and on the 31st he complained of pain at the bottom of the back and left side of chest. On the 31st 10 millions were again injected; the right leg was now painful and the thigh swollen. The next day another 10 millions were given, and pain was complained of in both thighs severe enough to require morphia. On the 3rd 15 millions were given, and on the 4th patient felt much better and the temperature dropped to normal. On the 5th and 6th 25 millions were injected. The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106°
105°
104°
103°
102°
101°
100°
99°
Normal
98°
97°
Temperature (Fahrenheit)
1st rigor
2nd rigor
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CHART 8.—Case 9.

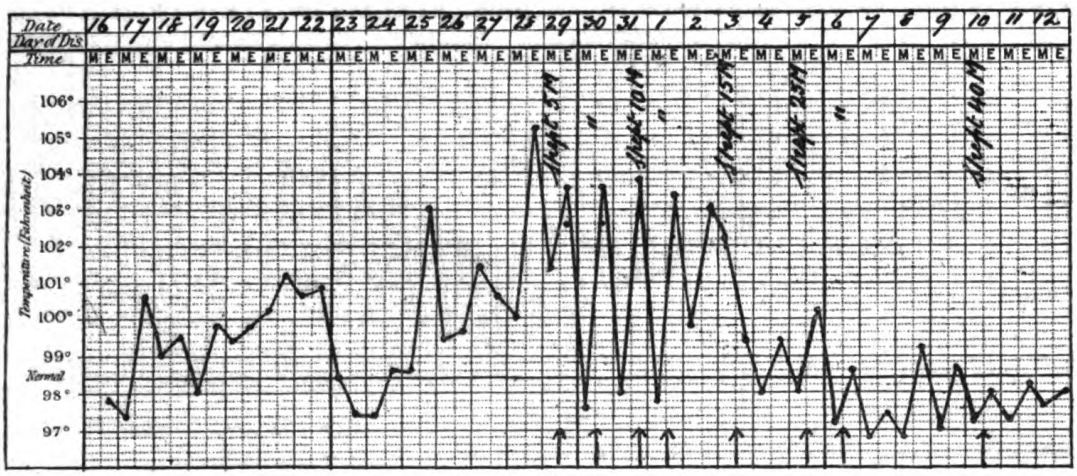

CHART 9.—Case 10.

swellings on the thighs and chest had by now disappeared. It was interesting to notice the rapid development of local areas, which threatened to suppurate, but did not do so.

CASE 11: *Staphylococcic septicæmia and* B. coli *pyelitis.*—A young man, æt. 23 years, under the care of Dr. C. Reid, of Stafford, suffered from colitis and jaundice in February, 1911. He apparently recovered, but in March was operated on by Mr. Barling for appendicitis. A much swollen appendix was found, with much free seropurulent fluid in the abdominal cavity. Peritonitis followed, and the wound took a long time to heal, but after a month the patient recovered sufficiently to be allowed to walk. At the end of April his temperature rose again and he became acutely ill. A specimen of his blood was sent to the Clinical Research Association, and a pure culture of *Staphylococcus aureus* was found. On May 18th and 21st 50 million staphylococcic vaccine were injected, but he became worse, and on the 30th had a severe rigor. On June 13th he was seen in consultation with Dr. Reid. He was then extremely wasted; there was a well-marked *risus sardonicus.* Mitral and pulmonary systolic murmurs were present, and the pulse was soft and small. At the base of the right lung there was a patch of dulness, the right hypochondrial region was rigid, and the right kidney palpable and tender. The leucocyte count was 17,500. The blood gave a pure culture of *Staphylococcus aureus.* The urine contained albumen, many pus-cells, and from it were grown *B. coli* and *Staphylococcus aureus.* Exploratory puncture of the right base resulted in a little blood-stained fluid from which also *B. coli* was grown. On June 18th a dose of 5 million staphylococci and 5 million *B. coli* was given. It then seemed impossible that the patient could live many days. The vaccine was given every other day and sometimes every day, the doses being gradually increased to 20 millions of each germ by the beginning of August. Improvement occurred slowly but surely, and coincided with the appearance of large abscesses, which developed with remarkable suddenness in the subcutaneous tissue of the legs and back. In September pus was passed *per rectum.* By the beginning of September patient was able to sit up a little each day. In November patient said he had not felt so well for years, but temperature still reached 99·5° F. most days. Vaccine treatment was stopped for a time at the beginning of June, 1912, but in August three small abscesses have appeared and vaccine was again given. Altogether more than seventy doses of vaccine have been given in doses ranging from 5 to 500 million staphylococci. The patient is now, April, 1913, quite well and has hunted since Christmas.

CASE 12: *Gonococcic septicæmia.*—A lad, æt. 20 years, was seen with Dr. Stuart Moore and Dr. Stacey Wilson on April 8th, 1911. Two months before he had a severe attack of gonorrhœa, followed by arthritis in both knees. Fourteen days before he became acutely ill, and his temperature, of an intermittent type, rose each day to 101° to 102° F. When seen on April 8th he was delirious, and at times almost maniacal. There was an

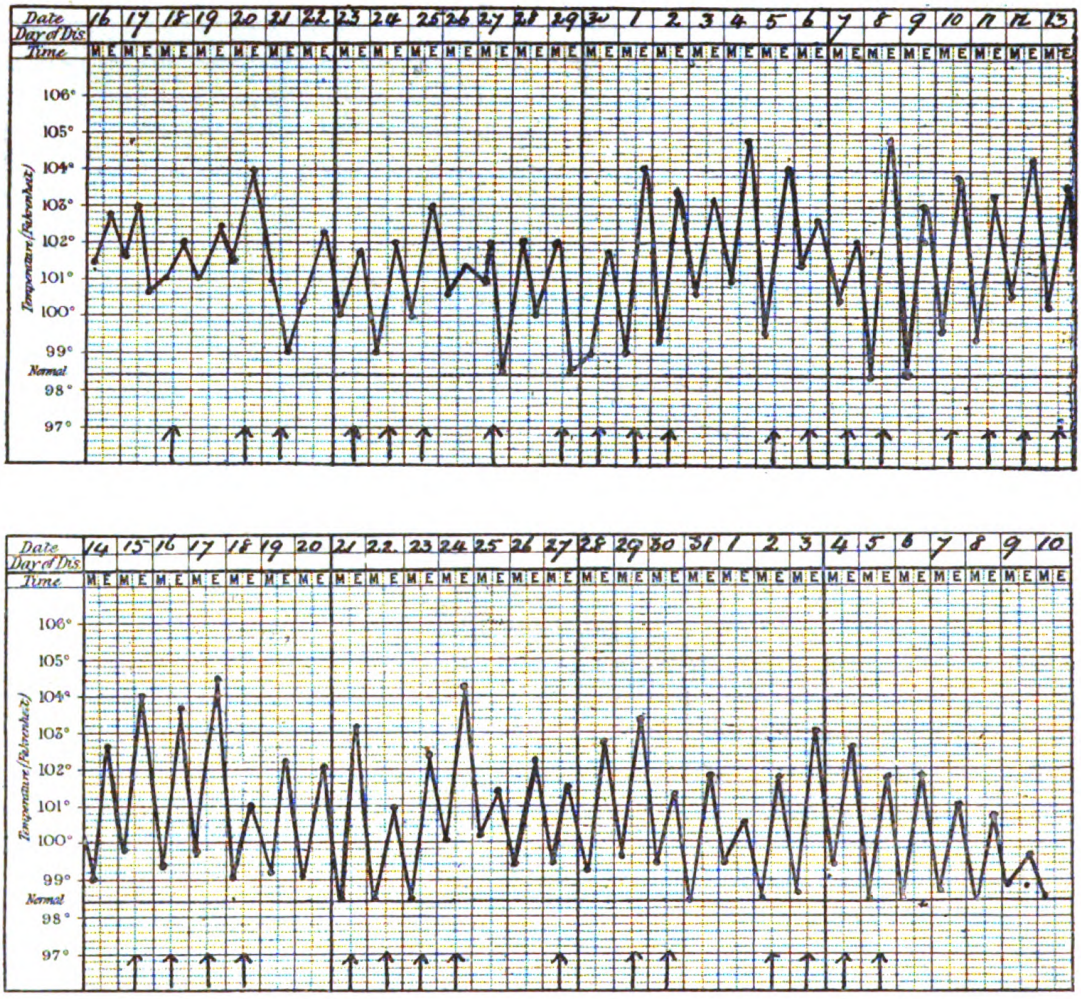

CHART 10.—Case 11.

irregular swelling in region of right kidney, and a loud mitral systolic murmur was present; there was much wasting; leucocytosis of 28,000. Blood-cultures were made and a pure growth of the gonococcus was obtained. From the urethral discharge the gonococcus could be seen in films, but cultures were unsuccessful. The urine contained pus-cells and *B. coli*. On April 11th 5 million gonococcic vaccine were injected, on the 14th 10 millions, and on the 18th 15 millions. The last dose of 20 millions was given on the 25th. The temperature improved for the three days

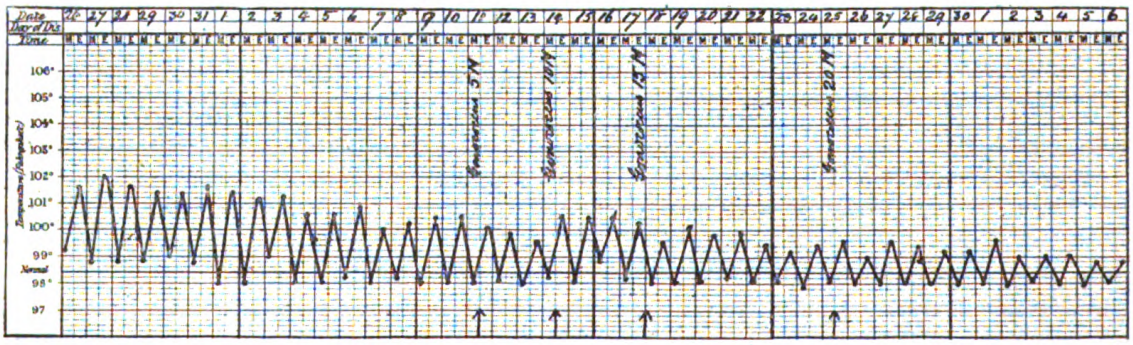

CHART 11.—Case 12.

following the first injection, the second injection was followed by a rise of temperature, but the third again by a fall. After the first injection the general condition began to improve, and there was no delirium after the 16th. Temperature continued to fall slowly and reached normal by May 2nd. The temperature chart shows no striking change as the result of the vaccine, but clinically the patient, who was getting worse up to the day of the first injection, afterwards began to improve, and this improvement continued without interruption.

CASE 13: *Gonococcic septicæmia.*—A young man developed septicæmia after an acute attack of gonorrhœa. He was seen with Dr. Welsh on April 10th, 1911. He had then been ill for a fortnight. On April 4th an embolus lodged at the junction of the right subclavian and brachial arteries, and when seen no pulse could be felt at the wrist. He was sweating profusely, had severe rigors, and there was a loud harsh mitral systolic murmur. His condition appeared quite hopeless. A dose of 5 million stock gonococci was at once given and blood-cultures taken, from which a pure growth of the gonococcus was obtained. On the 12th 10 millions of the autogenous vaccine were given; the pulse was then 130 and the urine contained much blood. On the 14th another 10 millions were given. Urine now scanty, and still contained much blood. On the 17th crepitations were now found at the base of the right lung, and expectoration was blood-stained. He was very restless, complained of tingling in left arm, but looked and felt better. On the 20th suppression of urine, ankles much swollen, and a purpuric rash appeared on the legs. There was also slight ascites. Much bright blood was coughed up from the lungs for last two days. Patient died on the 21st. No vaccine was given after the 14th, as his condition was quite hopeless.

Fifteen cases of infective endocarditis in which a streptococcus was isolated from the blood have been treated by vaccines, but in no case with success. In five no result seemed to follow the treatment, in two the patients distinctly became worse, in the other eight there was distinct improvement, but this was not maintained, and the patients died of cardiac failure or cerebral embolism.

Apart from the cases of ulcerative endocarditis, these records show that vaccines may be depended upon to give considerable help in septicæmia.

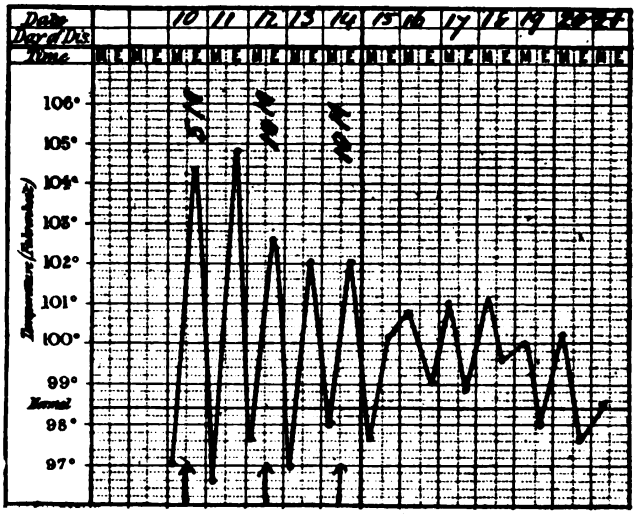

CHART 12.—Case 13.

The cases were all of great severity, and I think a much greater mortality might reasonably have been expected. Most of the temperature charts show an alteration in the type of temperature following the first or second dose of vaccine, the temperature, which was previously remittent or sustained at a high level, becoming intermittent. Associated with this change in the temperature, subcutaneous abscesses were often noticed to appear frequently with great suddenness. These might subside without suppuration. It is apparent that the change of temperature is evidence of an attempt to localise the infection, and marks the transition from a state of septicæmia to one of pyæmia. This I regard as a good sign, and if the first dose of vaccine does not bring about this change I believe it is well to increase the dose until the change occurs. In most of the cases sodium citrate in doses of half to one drachm was prescribed with the intention of preventing clotting. In cases of carbuncle and brawny inflammation I believe it to be of great value, but I cannot say that I have seen any advantage from its use in septicæmia.

In the earlier cases an attempt was made to control the dosage by estimations of the opsonic index, but even if the index were a true guide to the action of the vaccine, which I believe it is not, there remains the difficulty that by the time the index is estimated and the injection can be made the index will have changed. The temperature is the chief guide, and it should be recorded at least every four hours, but it is useful to have another chart showing either the highest temperature

reached each day, or the highest and lowest.* The ordinary morning and evening chart is quite useless. The vaccine should only be given at a time when the temperature is falling.

Every indication afforded by the pulse, respirations, the various symptoms, and especially the patient's subjective feelings must be carefully noted. The administration of the vaccine must be controlled by one who, besides the necessary knowledge of bacteriological technique, has the clinical experience which will enable him to make use of every clue afforded by minute changes in the patient's condition. He must be a physician as well as an immunisator.

April 28th, 1913.

* The above charts are of this kind.

A Simple Method of Treating Epistaxis.—Pech (*Gazette des Hopitaux*).—Epistaxis being due to the rupture of the capillary vessels of the nose owing to congestion of the mucous membrane, the treatment is to lower the arterial tension while favouring coagulation of the blood at the bleeding point by exposure to the air. Lowering of the cerebral blood-pressure by prolonged forced inspiratory efforts in the following manner fulfils both these indications. The patient obstructs the nostril, which does not bleed, by pressing the ala against the septum with the finger, sufficiently firmly to reduce the opposite nostril by which the blood escapes to a slit. A forced inspiration, lasting five to eight seconds, is then made through this nostril, the mouth being closed, and the chest, when distended, emptied by a short expiration through the mouth. This process is repeated twice or thrice, preferably in the standing position, with the head erect; but the fall in the cranial blood-pressure is so marked that to persist would produce syncope. Care must be taken not to detach the clot in the nostril and thus cause recurrence of the bleeding.

THE DIAGNOSIS AND TREATMENT OF SCOLIOSIS.*

By E. GILLESPIE, F.R.C.S.

Assistant Surgeon, The Prince of Wales General Hospital.

By the term "scoliosis" is to be understood a constant deviation of the spinal column, or a portion of it, to either side of the median line of the body, with resulting distortion of the trunk. But if we confine our attention to such constant deviations we render the task of treatment so much the heavier, because, realising the marked deformities only, the early stages are liable to escape notice, and valuable time be lost.

There are two sharply defined groups—(1) the congenital, (2) the acquired, the former resulting from causes in action in early embryonic life, due to errors in development of either bone, vertebral or costal, or of malposition *in utero*; the latter secondarily to some skeletal defect occurring after birth, or more simply as a disturbance of balance in the poise of the body during the period of active growth. In the latter group the age-incidence is commonly between ten to sixteen years.

The common change is a lateral deviation of the spine, with rotation of the vertebræ. By deviation is to be understood the dropping away of any part of the column from the mid-vertical line of the body as measured by a plumb-line; and by rotation the twisting of the body of a vertebra through a vertical axis in relation to its adjacent members in the series.

It is easy to picture the skeletal changes which occur as the result of such abnormal strains and pressures on the comparatively soft bones of the young. Experimentally, the changes have been produced by fixing the bodies of young dogs in definite positions by plaster-of-Paris jackets. In the acquired type the early changes are in the ligaments and muscles, the later in the bones and discs.

With regard to *diagnosis*, there are few conditions with which scoliosis can be confused. The more important differentiation is to find out under which of the many types of causation the case should be placed.

* A Post-Graduate Demonstration at the Prince of Wales's Hospital.

Examination.

The first essential is to have the patient properly prepared for examination. No half measures are to be taken. If a child, the whole body should be stripped, and the patient made to stand with bare feet on a table. In an adult the whole trunk is to be exposed to the level of the great trochanters. Below that level the parts may be covered by a light skirt, fitting closely at the top by means of a rubber band. Once more, the feet should be bare. It must be remembered that the legs are to be examined and measured. Unless in a case of doubt, the whole examination can be conducted from behind.

Next the position: The patient is directed to stand in the natural attitude with the arms hanging loosely by the sides. It is frequently of value to keep the patient standing for a few minutes before noting this posture, because the inclination of the patient is to hold the body stiffly when it is uncovered for examination. Gradually, as the shyness or feeling of strangeness passes away, the curves become apparent. Next mark out the bony prominences and the mid-vertical line with a plumb-line passing through the notch of the buttocks. Head suspension is useful to gauge the fixity of lateral deviation.

Again, with the knees straight, the patient is instructed to bend forward in the effort of trying to touch the toes; the observer then looks along the back; a difference in level of either side indicates rotation of the vertebræ. Flexibility of the spine is tested by seeing what amount of correction the patient can bring about voluntarily, as by altering the level of the limbs with blocks under one foot, etc.

Lastly, skiagrams should be taken from all cases. Their value may be illustrated by the plate which I hand round. You will see developmental defects not only in defective formation of the bodies of vertebræ in three regions, but also in the ribs, producing a complicated curve.

With regard to disease differentiation, care must be exercised to recognise the occasional cases both of Pott's spine and of scoliosis, which present very similar clinical pictures of pain and deformity. A few cases of Pott's curvature show a lateral deviation. But the deformity has been described as if the shoulders and upper part of spine were slipping down from the lower, rather than the bending and curving of a scoliosis. Again, in a few cases of scolicsis a distinct projection of the spinous processes is found at the point where two curves meet, and more especially if the curves are of nearly equal length. So you see that if pain from strain of ligaments and spasm of muscles be added to the picture, some cases of decided difficulty may arise and require repeated examination before an opinion can be expressed. Really no time will be lost, because in such cases the treatment is similar for both, namely, rest. Nor can much be expected from X-ray examination, because little or no change may have taken place in the bones.

The following list, although not exhaustive, gives some idea of the many conditions which may upset the balance of the spine and cause a scoliosis to appear.

Congenital.—(1) Congenital defects in the development of the vertebræ. Half a body may be absent in different regions and cause a complicated deformity.

(2) Congenital defects in the absence of ribs on one side.

(3) A type stated to be due to malposition *in utero*—of little clinical interest, because the subjects are usually non-viable.

Acquired.—(1) Constitutional.

(2) Result of laborious occupation.

(3) Empyema.

(4) Paralytic: { Infantile. / Spastic. / Syringomyelia. / Ischias scoliotica.

(5) Rickets.

(6) Curve associated with adenoids and nasal obstruction.

(7) Hysterical.

(8) Resulting from a healed morbus coxæ.

Treatment.

Let us suppose that the diagnosis has been made; the best plan for us in the time at our disposal is to take up a typical case and follow out the lines of treatment.

(1) *Congenital case showing bone defects.*—These cases are observed in very early years. The child should be kept flat on his or her back in a light wicker-work cage or on a flat back-board, the object being to avoid all abnormal pressures and

strains, such as are commonly produced by carrying the child in the arms.

Later, as the child begins to assert its inclination towards the upright posture and walking, a properly fitting brace or jacket must be fitted to carry the weight of the body from the shoulder-girdle to the pelvis. Naturally this support will require reasonable altering to meet the demands of growth.

The period of observation must extend over many years. The possibility of doing without a support must be gauged by trial. There is no necessity for massage, because the general condition of the patient is good and the muscles are developed. But suitably devised exercises are of value to promote proper balance in the spine.

Acquired rickets.—Probably many of the cases of scoliosis have their origin at as early a date as the time when the child begins to attempt to sit up, and pull is exercised upon the spinal column by the psoas muscles.

Here prevention is the best means. The same rules apply as to the previous type, but in addition there is a distinct disturbance of nutrition, and, consequently, a poor muscular condition which is to be combated.

It must be realised that there is no wonderful and powerful force at work to cause the deformity, but merely an alteration of direction in many small forces. Carrying the child in the arms and allowing the body to fall away from the nurse is a potent factor in producing the condition; another is the twisting and turning in the attempt to sit up.

In addition to the mechanical treatment, attention must be paid to the diet, and means taken to supply plenty of fresh air.

When the acute stage has passed away, passive movements to correct the deformity and massage to the spinal muscles should be used.

No fixed apparatus should be used in the early days of rickets. The children are so liable to intercurrent affections. The weight of a plaster-of-Paris jacket is too great; besides, these children perspire freely, and it is difficult to keep them clean.

Empyema.—If the empyema has been diagnosed and treated at an early date in the illness, no deformity should arise. The lung expands almost to the chest capacity, and there is no alteration in the balance of the spine.

It is far otherwise where the diagnosis has been difficult, or strangely enough, as sometimes happens, the patient has "a bit of a cough," and never sees a doctor until exhaustion forces his hand. The lung may never expand. Extensive operative measures may have to be employed before healing takes place.

The poor expansion of the lung and the loss of support from the ribs readily allow of deviating and distorting forces coming into play.

To realise that a scoliosis may occur is half the battle. By keeping the patient under observation and combining ordinary graduated gymnastic exercises with attempts to expand the lung, most cases can be prevented and kept under control.

If the patient comes under your notice with a fixed curve, the quickest and surest method is to employ a plaster-of-Paris jacket, and afterwards the routine of exercises to develop the muscles.

Paralytic.—Most resistant and fixed deformities arise from neglected cases of paralysis. Attempts must be made to mobilise the spine, as will be indicated when we consider the large group of postural and constitutional curves. No complete rectification can be expected and sometimes very little improvement after the most strenuous treatment. A properly fitted brace must always be worn. It is possible that operation may help in the severe cases.

Some authorities have tried to make a separate group of cases of scoliosis due to the effects of enlarged adenoids and tonsils. This seems to be unnecessary, since all the effects can be ascribed to their influence on the body and the mind. The curious mental apathy and inability to concentrate the attention have been fully described. These, with the deficient oxygenation of the body and the probability of septic infections, are surely predisposing factors and require no special grouping. The cure following treatment is merely the effect of improved general health.

Hysterical.—Occasionally after a careful examination one can exclude an organic lesion, and make a diagnosis of a hysterical scoliosis. One peculiarity in the appearance of the patient is the resemblance to a bird simulating a broken wing.

It is useless to tell the patient and the relatives that there is nothing the matter. They will merely point out the marked deformity—not noticing the exaggeration of it when attention is direct to the patient—and call in another doctor.

Once you can convince the friends that the

patient is able to control the deformity a good journey along the road of treatment has been made. The same effect may be gained by removing and controlling sources of sympathy, especially if it is the case of a child. A little discipline, application of the battery and vigorous massage will help all but the most obstinate cases.

In the latter it may be that there is a fair field for treatment by "suggestion."

These comparatively brief remarks clear the ground for a fuller consideration of the large group of constitutional scoliosis. There are two large groups to be considered: (1) those with deformity but no fixation of it; (2) those with deformity and fixation.

The former class can be very efficiently treated by exercises under supervision, after any defect such as a shortness of a leg has been allowed for and corrected. Defective vision and hearing must receive attention. The clothing should be so arranged that no weight is carried on the tops of the shoulders.

One absolute essential in carrying out the exercises is to arouse the interest of the patient, so that all movements are made purposeful and vigorously. Hence the necessity for efficient supervision. Frequently the patients are in a poor state of health, and are quite incapable of keeping up a sustained effort. Therefore graduation in duration as well as in the effort of the exercises is very necessary. Again, the observer can watch the back, and help greatly in the education of the muscles by pointing to a special group and asking the patient to make the muscles contract.

All the preliminaries of general health have received attention; then just as systematically as in the treatment of phthisis the day must be mapped out into periods of work and rest, the latter in the early days being many times longer than the former, in the nature of an experiment to test the powers of endurance of the patient. Some such scheme as the following may be used:

The day begins at 9 a.m. and finishes at 5 p.m. Breakfast at 9 a.m.; rest until 10 a.m.; exercise 10–10.20 a.m.; rest until 12 noon; exercise 12–12.15 p.m. Rest and midday meal until 2 p.m.; exercise 2–2.15 p.m.; rest until 4 p.m.; exercise 4–4.15 p.m. As the fitness improves, so the periods of exercise are increased at the expense of the rest until the portion devoted to gymnastics occupies between three and four hours.

Types of Exercises Illustrated.

I will now point out and demonstrate some of the typical exercises.

An important essential is to have proper fixation of the pelvis. It is too cumbersome to hold the patient while he is exercising. For a small outlay a suitable platform with a vertical post for fixation can be obtained.

The exercises should be simple in type, and should show a visible effect in straightening the spine. After the preliminary testing period the gymnastics should occupy one to three hours daily, and be carried out under supervision for six to eight weeks. Every effort should be made to avoid the irksomeness of routine work, by compelling the patient's interest. During the course the weight should be noted weekly, because of the important indication of the progress. After this period, if progress is satisfactory, it is sufficient if the patient reports at long intervals.

Symmetric exercises (demonstrated).—From the fundamental position of hands pressing well down upon the hips. Breathing regulated to the movements.

(1) Shoulder raising and sinking. Affects the lumbar region.

(2) Bending to the horizontal and back affects lumbar region.

(3) Trunk circling.

(4) Swimming movements.

Exercises in the horizontal position.—Head suspension: This is corrective through the whole length of the spine. The action is passive. It is a valuable mobilising procedure.

Asymmetric exercises.—(1) *Hip sinking*: Foot advanced on opposite side to the convexity. Acts on lumbar curve.

(2) Self correction: Hand on thorax on side of greater curve, other hand on ilium for dorsal or lumbar.

(3) Trunk hyper-extension with side bending for lumbar.

(4) Partial suspension: Arm and leg locked; suspension by other arm. Acts on dorsal curve and lumbar.

Creeping exercises.—The patient keeps the body in an approximately horizontal position, with

the hands and knees on the floor. The hands, knees and toes should be protected; only learned from practical demonstration. Scoliosis with fixation is much more difficult to treat, and the difficulty of obtaining a good result must always be borne in mind. Yet much may be attained in the direction of improved appearance, even although an anatomical restoration be impossible.

Efficient treatment is slow in effecting a result, and will try the temper of the medical man, the patient and the parents. In many early cases an attempt may first be made to improve the flexibility of the spine, but should no result be obtained after a few sittings, it is much better to proceed at once to forcible measures. Many elaborate forms of apparatus have been devised. Probably better results are obtained by side-pressure, in the horizontal posture, rather than by stretching in the erect. A very simple but powerful apparatus is as follows. The cost is low.

The patient lies face downward on a table (3 ft. wide by 4 ft. long) with the knees flexed.

A canvas strap passes round the thorax; it is fastened to a hook at the side. A similar one passes round the pelvis at the level of the trochanters and is fastened on the same side. A third strap is placed between the other two, so as to exert pressure and twist upon the maximum curve, but in the opposite direction. The pull is exerted by a cord from the strap passing through a compound pulley.

Perhaps one of the most useful methods of treatment in obstinate cases is the plaster jacket. Means should be taken to apply the plaster while connective pressures are in action. Extension can be combined with side-pressure. The patient is clad in a stockinette undervest. It should be loose so that it can be fastened below between the legs, and also pulled well up over the neck if it is necessary to enclose it in plaster. The pelvic portion must be made very strong. After the apparatus has set, the edges are carefully trimmed, and windows cut over the concave portions to allow expansion.

A word of warning must be spoken about the sudden abdominal distension which occasionally occurs. It is most distressing, and accompanied by alarming dyspnœa. It may be that the plaster must be cut away at once. I have only met one case, and in it there was a secondary deposit of carcinoma in the spine where I applied a jacket for support and to relieve pain.

The time that a jacket can be worn comfortably varies very much, and depends largely upon the cleanliness of the patient's home. About the average time is six weeks. The number of applications is to be judged by the benefit obtained.

Subsequently the same regular attention must be paid to gymnastics if a satisfactory maintenance of correction is to be preserved.

I have said nothing about prevention, but the measures for the routine examination of children at school make one hope that from the possibility o early treatment many, if not all, of the serious cases will disappear from view. My remarks are in no sense a full account of scoliosis, but merely to serve to direct your attention to a condition which is apt to escape the attention it deserves under the name of a "growing-out shoulder."

April 28th, 1913.

Toxic Effects of Neo-salvarsan.—A. Bayet, of Brussels, says that the necessity for retracting early statements follows premature publications as the shadow follows the solid body. But he yields to the request of the *Presse médicale belge* (1912, lxiv, 755), and reviews his experience with neo-salvarsan. He has found that the immediate disturbances following injection of neo-salvarsan are less than with salvarsan, but later there is little if any difference in the toxic action of the two drugs. Even in his brief experience (April to September) he has had two severe cases showing this toxic action. One was in a man of 24 years, with syphilis of three months' standing; 0·07 grm. of neo-salvarsan was injected and eleven days later 0·08 grm. Immediately fever and a scarlatinal eruption developed and he vomited for two days, but there was no headache. The fourth day these symptoms had subsided, but both legs and the bladder became totally paralysed, with other signs of profound injury of the nerve centres in the lumbar region, but nothing to indicate meningitis or polyneuritis, syphilitic paraplegia or a nerve relapse. The Wassermann test was negative. In the second case bilateral neuritis of the nerves of the forearm with atrophy of one interosseous muscle followed five injections in two weeks, amounting to a total of 2·5 grm. of neo-salvarsan.—*Journ. A.M.A.*, vol. lx, p. 919.

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SOME REMARKS ON THE DIAGNOSIS OF PRIMARY INTRA-THORACIC MALIGNANT DISEASE.*

By W. H. MAXWELL TELLING, M.D., B.S.Lond., M.R.C.P.Lond.,
Physician to the Leeds General Infirmary.

SOME cases which I have recently seen have recalled certain experiences in connection with malignant disease in the chest and the difficulties such cases seem to present to many observers. Excuse may be found for the following remarks if they serve to direct attention to certain broad features of these cases, which may help to make their diagnosis less difficult, perhaps mainly because they may lead to a closer study of them.

The first important point which I want to make is that these cases are not of such extreme rarity as is often supposed; any practitioner of large experience may easily have seen a number of such cases. My house-physician, Mr. Dunbar, has looked up the cases coming to post-mortem examination in the Leeds Infirmary during the years 1900 to 1910 and has collected 41 cases. The majority of patients, realising the incurable nature of their disease, prefer to die at home, and when one adds these to the number its magnitude supports my contention.

In private practice these cases are those in which a consultation is sought sooner or later when means allow, and I have always been impressed by the fact that few chronic cases seem to give the practitioner so much all-round difficulty in diagnosis. And some of the reasons are not far to seek. In the first place, I think many observers, believing in the absolute rarity of the condition,

* Read before the Leeds and West Riding Medico-Chirurgical Society.

are "taken by surprise," so to speak. If in every case of intra-thoracic disease which presented unusual features the possibility of neoplasm was remembered, I have not the slightest hesitation in saying that not only would a number of cases be reclaimed from the limbo of erroneous diagnoses, but that the diagnosis itself in a great proportion would be found to present no special inherent difficulty.

In the brief space at my disposal I can only hope to act as a sort of sign-post on the road which should be travelled in the systematic study of the cases. The inclusive nature of my title—all primary malignant neoplasms in the thorax (but in particular those causing respiratory symptoms)—would effectually preclude any detail. Nor is there necessity for much detail. Once one has one's suspicions aroused as to this possible nature of the case reference may be made to the detailed accounts excellently given in many text-books, but not in all. Some are far too scant in the treatment of the subject, while it has seemed to me that others so confuse the broader aspects of the cases by the introduction of the many and great pathological uncertainties, or supply so many facts, and in such a disjointed way, that all true perspective is lost, and these chapters are either avoided altogether or their true significance is largely missed.

In a good text-book, as, for example, Allbutt's 'System,' the various types are described under at least four headings in different parts of the work. In each case the descriptions are good and very full, but unfortunately patients—especially obscure cases—do not come to us labelled and sorted out, so that we can look up difficult points in a particular chapter with the certainty of enlightenment. They come to us rather with certain slight or vague symptoms more or less directly referable to the thoracic viscera, more particularly the respiratory viscera, and we have to start from that end of the problem. And even if we get on the track of malignant disease, and correctly withal, we still desire to make out its exact localisation and pathological nature. It is therefore necessary to be able to envisage the broad clinical aspects of all these types taken together, and then by a close and very profitable clinical study we may correctly work out the details and diagnosis in the majority of them. The great thing in the first place is to determine that we have to deal with cases of intra-thoracic neoplasm and not with anomalous cases of, say, tuberculosis. It may be a matter of great uncertainty to say whether the lesion is of pulmonary or mediastinal origin, but that after all is of but secondary importance. In no class of case is it, therefore, so important to have a sound working knowledge of the general lines of diagnosis, and, so far as my personal experience goes, in no class of case does this seem so difficult to acquire.

So I have designedly lumped together all the varieties of intra-thoracic malignant disease tending to cause respiratory symptoms—carcinoma, sarcoma, endothelioma and other rarer varieties—and likewise have included all the principal foci of origin—mediastinum, bronchi, lung and pleura, as well as other situations—so that we may view the matter from the position which is so seldom defined in the text-books; that of being confronted by a patient who has symptoms pointing to *some* disease *somewhere*, and with the task of puzzling it all out as best we may. Text-books for the most part describe diseases which we are left to fit to the patients: in practice we get the patient first and have to find out which disease he can be fitted to.

The basis of all sound clinical diagnosis is a rough but accurate knowledge of morbid anatomy, and I think the point of first importance in understanding these tumours is a general knowledge of their commoner sites of origin and of their directions and methods of extension. From this it is easy to appreciate the structures that are likely to be secondarily involved, and with knowledge of the known tendencies of malignant disease to infiltrate, ulcerate, undergo secondary degeneration or infection, and in some cases contraction, from the presence of much fibrous tissue, it becomes fairly easy to understand and correlate the various symptoms, and to understand some of the very various guises in which this form of disease may present itself.

The principal places of origin are:

(1) The mediastinum, anterior or posterior.
(2) The bronchi.
(3) The lung-tissue.
(4) The pleura.

There are others: the bones, the œsophagus (a special class of case which seldom causes difficulty), the heart, etc., but they need not specially occupy us.

Mediastinum.—Here tumours of glandular origin

are commonest, and this group of cases presents fewest difficulties in diagnosis. The essential fact is an enlarging tumour in a small space which is crowded with important structures, which may become displaced, compressed, or actually eroded. Certain forms, notably the relatively common lympho-sarcoma, tend to enwrap and compress the structures without actually invading them. Clearly, therefore, this mediastinal group will give early sign of the presence of disease, frequently by irritation of the bronchi or trachea, or by narrowing these structures and producing stridor. Further common results are bronchial stenosis or occlusion; pressure on the great veins with cyanosis and œdema of the upper half of the body; pressure on nerves, especially the recurrent laryngeal, causing hoarseness, aphonia or dyspnœa from spasm; pressure on the œsophagus with dysphagia; displacement of the heart, invasion of the pericardium with possible pericarditis; extension to the pleura with frequent pleurisy and effusion; infiltration of the chest-wall and especially secondary deposit in accessible glands, above the clavicle or in the axilla: these are all such obvious possible results as immediately to arouse suspicion when they are encountered.

The cases of pulmonary origin are more difficult. There are two main groups—those in which the growth originates from the bronchi (these are generally and usefully included in the group of cases of malignant disease of the lung), and those arising in the parenchyma.

The classical and commonest type of lung cancer is that arising from a main bronchus or one of its main subdivisions, and therefore arises at or near the root of the lung. Effects are produced (*a*) by the mode of growth at the focus of origin, and (*b*) by the extension of growth from that primary focus. Locally these growths tend to form tumours of varying size. A very constant tendency is to interfere with the normal lumen of the bronchus; they tend to narrow it, which may be revealed by stridor, or by the occurrence of bronchiectasis in the tributary area, with the signs and symptoms of that condition. In rare cases total occlusion, with atelectasis of the lung, results. Fungation into the bronchus may occur, causing much cough and irritation, bronchitis, and, not infrequently, hæmorrhage.

But the mode of extension of this type of cancer at the hilus is important if one is to understand some of the more puzzling clinical manifestations of cancer of the lung. Frequently the local tumour formation is slight but the growth spreads—by vessels, nerves or bronchi—into the pulmonary tissue, so that we have a mass radiating centrifugally from the root of the lung. If the growth is chronic and fibrous to any great extent (and this is frequently the case), we get airlessness and immobilisation of the affected lung, and may even get *retraction* of the chest-wall from the contraction of the lung. The possible clinical phenomena resulting from this mode of extension, often complicated by more or less tubal dilatation, are many and obvious.

But the growth may extend centripetally and invade the mediastinum, and frequently gives rise to metastatic deposit in the mediastinal glands. In some cases in which the primary focus remains relatively small and in which the mediastinal metastasis is relatively large it is clear that the cases will approximate clinically to the mediastinal type.

But the bronchial origin may not be at the root, but may be in one of the minor divisions more or less remote from the hilus. We get, however, the same centrifugal infiltrating tendency, but affecting a more circumscribed area of lung, and, though bronchiectasis often occurs, the resulting lesion tends to approximate more to the nodular type shortly to be described. Clearly in these cases one would expect the respiratory symptoms to be generally longer delayed and less severe. Certain other modifications will easily occur. In the first place great retraction of the chest-wall, extensive airlessness and immobilisation will be less frequent. But the pleura will be reached earlier. This may lead to an important masking of the disease, for with comparative latency of symptoms due to the actual growth, aggressive symptoms and signs of pleurisy and effusion may easily be caused, as well as the possibility of the rarer complications of empyema or pneumothorax.

And as the pleura is reached earlier the mediastinum tends to be invaded later and to a less degree.

The next anatomical group is the *nodular*—masses of cancer surrounded by lung-tissue—and of this there are three sub-groups:

(1) The latent.

(2) The multiple.

(3) The massive.

Whether these growths arise *from* the lung parenchyma or merely occur in it need not detain us now.

In the latent group we have a nodule of growth deeply embedded in the lung and giving rise to few, if any, symptoms, and to but slight signs to which attention is seldom drawn. They may be discovered unexpectedly post-mortem—I have seen one such case—or death may result from mediastinal or other metastasis.

The multiple group is the commonest. There is a primary nodule somewhere in the lung with perhaps satellite nodules round it and numerous metastases throughout both lungs, though such deposits are usually more numerous in the lung primarily affected.

Clearly this is a difficult group. The clinical picture will depend on the number of the deposits and the closeness of aggregation; on their rate of growth; on the occurrence of secondary necrotic changes in them, with possible septic absorption, or on such acute complications as broncho-pneumonia or even gangrene. And added to this is the frequent invasion of the pleura with the usual results of such invasion, or the occasional pressure or invasion of other structures such as bronchus or pericardium. Symptoms may be many or few, and very various.

The massive cases, though not common, are important. They are usually sarcomatous, starting from the root of the lung, but this origin may easily become obscured. They may attain a large size, and may be covered by just a shell of lung-tissue. Apart from necrotic changes, which may transform the picture into one of abscess or gangrene merely, the main effects will be due to displacement. In particular the heart will be pushed over and the chest-wall bulged. The physical signs of solid tumour are usually fairly definite and the diagnosis not of very great difficulty.

The last group of intra-thoracic growths which I propose to enumerate is that arising from the pleura, and cases arising from the chest-wall are usually indistinguishable from them. Primary pleural tumours are infrequent, though from what has been said before, secondary invasion of the pleura is relatively common. Their mode of growth is very various; it may be in the form of flattened growths over the surface of the lung, or it may be in one gristly cap, leading to compression of the whole lung and frequently to contraction of the chest-wall as the result of adhesions between it and the lung. The symptoms may be slight, and the signs will depend largely on whether adhesions or effusion takes place.

This is an incomplete, but I hope fairly useful summary of the anatomical possibilities of these growths. It is not difficult to grasp this amount of morbid anatomy, but until it is done an observer can have no really clear conception of what may be going on in the chest.

I next want to advert to a few of the more striking and important general symptoms, which should lead to our suspicions being aroused as to the possible nature of the case, or to such as are of especial value in diagnosis.

In doing this I desire to be very brief, and therefore shall not make any detailed reference to many of the pressure symptoms that arise so frequently in the mediastinal cases. These are so striking that the occurrence of *any one* of them should lead to an examination for possible growth. But I should like in passing to emphasise the importance of noting and investigating two symptoms that frequently result from such pressure: firstly, alterations of voice, and secondly, cyanosis and œdema of the face, neck or upper part of the trunk. They are both frequent, and pressure phenomena in general are usually so striking that when present no case of intra-thoracic neoplasm should escape detection for the mere want of being remembered.

The symptoms which do merit a little detailed explanation are pain, cough, sputum (including hæmoptysis), respiratory difficulties (including stridor), cachexia, and fever.

Pain.—This is a frequent though very variable symptom. From anatomical considerations it must be evident that many cases are likely to be free from pain in their earlier stages or for a considerable part of their course; sometimes pain is absent throughout. The pain is more frequently a dull ache rather than acute and lancinating, though all varieties are observed, sometimes in the progress of the same case. Referred pain is common, and its frequency should not be lost sight of. Pleuritic pain is frequent and requires no special comment. But no case of persistent pain, however slight, should be allowed to exist anywhere in the body without

careful physical examination; the case I am showing to-night is an excellent illustration of this.

Cough.—This is frequent, and owes its origin to various causes and is of very varying character. Though some cough is present in the majority of cases, its absence in all but the late stages is of sufficient frequency not to prejudice one against the diagnosis of malignant disease. It is often due to pressure on the trachea or irritation of nerves, and hence one may say that a purely irritative, non-productive cough should excite great suspicion of this origin. Frequently enough the cough is purely catarrhal, as bronchitis is a frequent concomitant. Generally, while cough is almost constant in phthisis, its absence in growth of the lung is not very unusual.

Sputum, including hæmoptysis.—Hæmoptysis is a frequent and suggestive symptom. It is not often present in the more easily diagnosed mediastinal cases, but in the lung cases its occurrence is of great value. It may be repeated and profuse or only slight: its occurrence with hard coughing has no more value in these cases than in other conditions. Much stress has been laid on the occurrence of altered blood in the sputum—notably the so-called prune-juice sputum. It is not pathognomonic, but when it occurs is extremely suggestive of the presence of cancer—only it very seldom occurs; I think with much less frequency than some of the books would lead us to suppose.

It is unnecessary to say that the sputum should in all cases be examined, but it is important to emphasise that *repeated* examination be made. Occasionally the diagnosis can be made by this means, but the cellular contents justifying this are a matter for expert pathological opinion, and then are liable to be much quarrelled about. Reference should be made to the literature for exact details of the possible morbid appearances of the sputum.

Respiratory difficulties, including stridor.—"Respiratory difficulties" perhaps sounds a little vague, but it is intended to be an inclusive term for all the forms and degrees of dyspnœa or alterations of the respiratory rhythm which may, and very frequently do, occur. I want particularly to emphasise the occurrence of *dyspnœa* as an early and suggestive symptom. It is frequently slight and insidious in its onset, and may be intermittent, perhaps only occurring on exertion. A characteristic point is its occurrence out of proportion to any discoverable physical cause; this is extremely suspicious. It may become very severe and paroxysmal; sometimes it is *definitely asthmatic* in type. The respiration may be obviously laboured or persistently quickened.

Any hint of stridor or stridulous breathing is a sign of the greatest moment. There are other causes of stridor, of course—aneurysm and syphilis of the trachea, for instance—but it is frequently present as a comparatively early sign in mediastinal cases or peribronchial growths. I have been struck more than once or twice by the way in which this sign has failed to be credited with its due importance; I think perhaps because it has been mistaken for bronchitic wheezing.

Cachexia.—On the whole cachexia is not nearly so striking a feature of thoracic as of abdominal malignant disease. The effects of thoracic neoplasms are so much more mechanical for a long part of their course, and pain is seldom severe, that this fact is not to be wondered at. When secondary necroses and septic complications ensue the cachexia may be rapid and severe, as also if there is extensive metastasis. In any case with marked respiratory symptoms and signs the comparative absence of cachexia is at any rate a suggestive point.

Fever.—Most cases are afebrile except when septic complications arise. The pleurisies and pericarditis cases are frequently without pyrexia, though notable exceptions occur. Some cases of massive growth are accompanied by fever, apart from such complications; this is a phenomenon observed in cancer elsewhere, but speaking generally, the absence of fever is a valuable point in differential diagnosis.

Duration.—This varies greatly, the limits being, perhaps, two months to five years. The acute cases call for less emphasis than the very chronic ones: when the duration of symptoms runs into years, it is not unnatural to begin to doubt the presence of malignant disease. But numerous extremely chronic cases are on record.

The physical examination in these cases is usually a fascinating study, but I must forbear to do anything more than emphasise a few general points of outstanding value. In describing the morbid anatomy frequent reference was made to bronchiectasis, atelectasis, solid lung, etc., and one must premise a fairly accurate knowledge of the varying

physical signs of these conditions, as well as a ready application in technique of examination, to enable a diagnosis to be made in a large number of cases. That is why they merit and repay careful study. There is no refinement of auscultation or percussion that may not be usefully employed, and the results obtained by such delicate technique may provide the deciding datum in diagnosis.

I want to emphasise the valuable information that may be gained from inspection alone, and I hope I shall not be regarded as impertinent if I suggest that this should be done on all occasions in a leisurely way and a good light.

Alterations in the shape or volume of the chest, especially on one side, are of the greatest importance. It is scarcely necessary to say that evidences of surface or infiltrating growth or marked local bulging could not possibly escape notice or correct appraisement, but the finer differences are equally noteworthy and far more interesting. In particular those cases associated with contraction of the diseased side should always be remembered. Impairment of movement is common, and, though usually accompanying alteration of shape, is of frequent occurrence without it; it is a sign of much importance. Another very important sign is the presence of enlarged veins on the surface, or in the neck, or down the arms. Such venous enlargement may be slight, though definite and variable. When enlarged veins exist, the presumption of growth is very strong indeed, generally with mediastinal localisation. Enlarged glands are sometimes *visible* in the supra-clavicular spaces, but more usually must be felt for in the earlier stages, either there or in the axillæ. A positive finding goes a long way to establishing the diagnosis. They are often overlooked, because the possibility of their existence is not remembered.

There I regretfully leave the physical examination, and, in conclusion, can only hope to adumbrate a few of the chief or more striking clinical presentations of these cases.

It is safe to say that whenever a previously healthy person develops marked respiratory symptoms or signs, which cannot otherwise readily be accounted for, the possibility of neoplasm should be seriously considered, whatever the age of the patient; for sarcomata occur in young subjects and mediastinal lympho-sarcomata in children with some frequency. The diagnosis of senile phthisis is one which I am certain conceals the true nature of a number of cases of malignant disease of the lung. Serious progressive disease occurring in a previously healthy subject, therefore, should arouse the gravest suspicion. Physical signs of an anomalous type or unusual localisation should in many instances raise the possibility of the presence of malignant disease.

There are two modes of onset that require special mention. The first is the insidious one, suggesting, perhaps, an ordinary case of bronchitis or emphysema, and no rule can be laid down for the detection of such cases. Two great safeguards exist, however: the fact that one is always suspicious concerning these conditions when the previous medical history does not lead one to expect their occurrence; and the second great safeguard lies in repeated careful examination of the chest. Perhaps the greatest pitfall is reliance for an unduly long period upon an original careful examination in which no positive findings were made; the examination in chest cases should be of some frequency.

The other deceptive mode of onset is by an acute inflammatory lesion—some cases present themselves as a pneumonia in the first place—but there again, while the diagnosis is obviously impossible in the acute stage, the axiom that a chronic chest lesion should excite suspicion of growth should prove helpful.

I have not thought it useful to encumber this communication by a description of acute miliary carcinomatosis of the lungs—a practically undiagnosable condition.

The cases with a "pleural aspect," so to speak, are numerous and important. A repeated pleural effusion is suspicious, and if that effusion is accompanied by little or no pyrexia, is blood-stained, or becomes so after a few tappings, or when aspirated fails to result in restoration of a previously displaced heart, or materially to relieve the respiratory embarrassment—this is a notable point—or reaccumulates with unexpected rapidity, then, in all these cases at least *suspect* growth. Cytological examination of the aspirate may be more helpful than in the case of the sputum.

Lastly there must be remembered the cases of pericarditis, or pericardial effusion; the occasional empyemata or a possible pneumothorax; erosion of the spine with possible cord symptoms; essen-

tially œsophageal cases; and lastly, and of great interest, certain cases of abscess and gangrene of the lung which have malignant disease concealed as a cause. It is tempting to refer to cases illustrating most of these points, but it would be outside the spirit of this paper were I to do so; my object has been the outlining of *principles* of investigation and the grounds on which suspicion should reasonably arise, rather than to furnish a welter of clinical material, howsoever interesting.

One point I must make, and that is that in every case in which there is reasonable ground for suspicion an X-ray examination of the chest should be made. Negative results will occur even when malignant disease is present, and too much stress should not be laid on such negative results. Many observers have been pessimistic of the value of X-ray examinations in these cases, but I have received such valuable confirmatory evidence and help in differential diagnosis that I now do not omit to have it done in every case in hospital practice and, whenever possible, in private practice. It should never be the preliminary examination, however, but only after a careful study and examination of the case has been made by what I am almost tempted to call the "old-fashioned" methods of physical examination—old-fashioned, at any rate, in these days when we so readily send the patient to the X-ray room, or some of his excreta or excised portion of his anatomy to the overburdened pathologist.

To sum up: my objects have been to controvert the supposed great infrequency of these cases, to suggest that they are interesting and informing cases for clinical study, and, while leaving practically all details on one side, to endeavour to convey some general working idea of the ways in which these cases may appear before us, in the hope that earlier diagnosis may occasionally result. This is not quite without practical bearing, for there is possibly some field, if only a restricted one, for the employment of surgery in malignant tumours of the thorax, whether for radical operation, or merely to relieve the pressure in one of the most distressing types of case a medical man is called upon to witness. A little has already been done in this direction, and it behoves clinicians to perfect their diagnosis so as to give surgery every chance. And if these fragmentary remarks should lead to the correct occasional replacement of a diagnosis of, say, the ubiquitous tuberculosis in some of the more obscure cases, I think time will not have been entirely wasted in thus reviewing the subject.

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SOME POINTS IN THE CAUSATION AND TREATMENT OF GASTRIC HYPERACIDITY.*

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THE difficulties and uncertainties which still surround the scientific classification of the disorders of gastric digestion must be my excuse for asking you to consider this evening a few points connected with those derangements of function which are connected with a single symptom, namely, increased acidity in the gastric contents, and more particularly increased amounts of hydrochloric acid. In an organ whose functions are manifold it is, I know, impossible to make any one of these a basis for pathological classification, but clinically the consideration of a single symptom or physical sign is often of value, as it enables the mind of the observer to connect up various chains of thought, and thus by a process of exclusion and deduction arrive at some sort of basis for the diagnosis and treatment of a particular case.

In the first place I should like to review very briefly a few points concerning the normal secretion of hydrochloric acid by the stomach. In man the total acidity of the "psychic" gastric juice varies to a certain extent with the character of the food ingested, just as does the digestive power and the total quantity of the secretion. The second "hormonic" outpour of gastric juice, the chemical reflex for which is started by the presence of food in the pyloric antrum, being smaller in amount than the first, alterations in the composition of the juice are more likely to depend on conditions influencing the former than the latter stimulus.

The total secretion of gastric juice has been estimated at about three pints in twenty-four hours with ordinary diets, and the percentage of hydrochloric acid at from 0·1 to 0·3. Clinically the total acidity and the percentage of hydrochloric acid is generally estimated at the height of digestion and calculated for the fluid contents of the stomach.

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One hour after an Ewald test-meal, rather less than half the total hydrochloric acid is already combined with protein in most normal persons.

Considerable variations may occur in persons showing no symptoms of digestive disturbance, both in the total acidity and in the percentage of free hydrochloric acid. Thus the total acidity expressed in cubic centimetres of decinormal soda solution may vary between 30 and 75, and the free hydrochloric acid between 0·07 and 0·2 per cent. The volatile acids also vary, and though usually equal to from 2 to 5 c.c. of decinormal soda, may rise to 11 c.c. (Harley). In persons over fifty years of age the free hydrochloric acid may be very deficient. Seiderlin and Liefschutz found it was absent in 40 per cent. of the cases one hour after the test breakfast.

While, however, considerable individual variations occur, it is remarkable that for any given person the percentage of hydrochloric acid is very fairly constant.

The normal limits being so wide, the question naturally arises as to the point at which hyperchlorhydria or excessive secretion may be said to occur. Theoretically a general statement is difficult to make; practically hyperchlorhydria is assumed to exist in certain clinical conditions which show a high *average* secretion where a series of cases is tested. Again, some difficulty may be found theoretically in determining between a positive and relative excess of hydrochloric acid, and at least one recent authority denies that a pure primary hyperchlorhydria exists (Bickel), and believes that all cases of hyperchlorhydria are really cases of super-secretion of gastric juice, with an absolute, not relative, excess of hydrochloric acid. Clinical observation on the whole, however, has distinguished between the cases of excessive secretion and those of relative excess of hydrochloric acid.

Hyperchlorhydria may occur in connection with gross organic lesions of the stomach and duodenum and also in cases where this cannot be shown to exist. Thus, in the former group, it is well known to accompany gastric and duodenal ulceration. In acute ulcer the proportion showing hyperacidity has been estimated at about one third, and in chronic ulcer at about one half of the cases. But though the total acidity in these cases may reach a point much above the normal limit, the free hydrochloric acid is not often above what may be normally found in certain individuals.

In another group of cases reflex irritation from lesions in some other part of the digestive system may give rise either to excess of hydrochloric acid or to true hypersecretion. Hyperchlorhydria may occur in atonic conditions of the stomach-walls. Lastly, in many functional and organic diseases of the central nervous system there is an occasional or constant high percentage of hydrochloric acid. This group of cases is of interest in connection with the preponderance of the nervous reflex over the chemical reflex in gastric secretion.

There remains what is perhaps the most interesting group of all, namely the so-called primary cases, which have been divided into two classes, namely, those in which the increased secretion of hydrochloric acid is due to inappropriate diet, and those in which it is caused by psychic or emotional disturbances.

Cases of primary hyperchlorhydria have now been so frequently described, that it will be unnecessary for me to enumerate their clinical symptoms or to describe their course. One point only must be mentioned—that is, the increasing number of cases which are now ascribed to some definite organic lesion, but which formerly would have been placed in the primary group. Surgeons especially regard primary hyperchlorhydria with considerable suspicion, and no doubt the tendency is to reduce this group to much smaller dimensions than formerly. However, it has not yet been completely extinguished as a clinical entity, and therefore it may still be allowable to discuss a few points which seem to be of interest in this connection. In the first place, the subjective symptoms (pain, etc.) bear no relation to the excess of free hydrochloric acid or to the total acidity found after a test-meal; in fact, the clinical picture of hyperchlorhydria may exist with a normal or subnormal acidity. "Larval" forms have also been described in which the excess of acid juice is rapidly poured out and then diluted by a neutral or alkaline transudation from the gastric blood-vessels. Schüler estimates these cases as constituting 20 per cent. of all cases of clinical hyperacidity. They can be detected chemically by withdrawing the stomach contents half an hour after the test-meal, or by taking the specific gravity and freezing-point of the gastric contents. Both will be found lower than normal.

In some cases, treatment on the usual medical lines by alkalies reduces the acidity, but in a large number the subjective symptoms disappear while the hyperacidity remains constant. There is evidence that in cases where the operation of gastro-enterostomy has been performed a diminution of the hyperacidity usually occurs. This was shown to be the case in twelve instances reported by Schur, and in five reported by Wilcox. In three of the latter series free hydrochloric acid was completely absent. If, as Wilcox suggests, this is due to regurgitation of alkaline substances through the artificial opening, it is clear that the effect is merely mechanical, and not due to any change in the secretory activity of the stomach consequent on the operation.

We are probably, therefore, justified in believing with Zweig and others that a diminution in the hydrochloric acid secretion is very unusual as the result of treatment, although a clinical cure may be accomplished; and this is certainly in accord with the generally accepted observation that in health the percentage of hydrochloric acid in the gastric juice is remarkably constant for any given individual.

The subjective symptom of pain, therefore, cannot be attributed solely to the presence of an abnormally large amount of acid in the stomach; some other factor must exist. A simple suggestion was that the gastric mucosa was over-sensitive to acid and responded by a painful sensation. The experiments of Hertz, however, go to show that the gastric mucosa is very insensitive to pain-producing stimuli, and that pain occurs only when the viscus is unduly distended. He believes that the increased acidity of the gastric contents, acting as a stimulus to peristalsis, causes excessively powerful peristaltic waves in the musculature of the stomach, and a consequent rise of intra-gastric tension, especially at the pyloric end. That some connection exists between excess of hydrochloric acid and pain seems to be shown by Friedenwald's observation that in ulcer, pain was more frequent where the acidity was high, and the further association of vomiting with high acidity, which he also noted, may be taken to corroborate the view that in such conditions an increase of intra-gastric pressure occurs.

The cases in which pain occurs with a relatively low acidity, however, require some further explanation, as does the maintenance of high acidity after the pain has been abolished. We are therefore again thrown back on the nervous apparatus of the stomach, and must assume that under some conditions peristalsis is stimulated to a greater extent by a given concentration of acid, with the consequent production of over-distension of the viscus and pain. Thus primary hyperchlorhydria would be, not a secretory neurosis, but a super-sensitive condition of the nervous mechanism, causing it to respond too violently to the stimuli applied by the gastric juice. This view, however, would not explain why in cases of ulcer pain should be more usually associated with high acidity, whereas this relation does not appear to hold good in simple hyperchlorhydria, or why, if vomiting is more frequent in ulcer with hyperacidity, it should be so infrequent a condition in the other class of case.

Spasm of the pylorus may in some cases contribute to the production of pain, but there does not seem to be sufficient evidence to show that this condition is at all frequently the result of hyperacidity alone. It is most frequently the result of a local lesion, and may then cause hyperacidity or super-secretion by producing stasis of food in the stomach. This of course could not be classed as a primary hyperchlorhydria. Moreover, clinically, the final results of hyperacidity are gastric atony and enteroptosis, or chronic gastritis with or without erosions, which are not conditions *primâ facie* likely to be associated with pyloric spasm as cause and effect.

I will now pass on to consider a few points in the treatment of hyperchlorhydria, which naturally divides itself into two headings—diet and drugs.

There can be no doubt that severe cases require a milk diet, modified with egg albumen and cream, which can be gradually increased by the addition of predigested carbohydrates until a mixed diet is tolerated. Many of these cases, according to modern views, are really cases of ulcer or some other organic lesion, and the treatment therefore will only be permanently successful when the cause of the hyperchlorhydria has been discovered and removed.

In less severe cases the question arises as to whether or not proteins or starches should be excluded from the diet. It has been shown by Meyer that in a healthy man with a high gastric acidity neither milk diet, meat diet nor vegetable

diet influenced the total acidity in the least. On the other hand, various observers have reported cases showing that the subjective symptom of pain can be influenced by the exclusion of one of the proximate principles of diet. No doubt in some cases the results have been due to the restrictions in the quantity of the food taken, and in others to the substitution of a more bland for a more irritating form of food apart from the chemical composition. Sörenson and Metzger devised test-meals on a protein, a carbohydrate and a mixed basis, having approximately the same caloric value and the same bulk and physical characters. Both protein and carbohydrate meals gave about the same amount of free hydrochloric acid. There seems, therefore, no experimental evidence for the plan of excluding proteins, which have the advantage of combining with some of the excess of free hydrochloric acid, and also of improving the nutrition of the patient. In fact the diminution of the amount of free mineral acid seems to be the important point; alkalies, which relieve pain, can only act in this way, as they have no influence on the acid phosphates or the organically combined hydrochloric acid—two other factors in the total acidity. Fats, especially in the form of cream, are able to inhibit gastric secretion to a large extent, but unfortunately many forms of fat are extremely distasteful to a large number of persons. Carbohydrates may be added to the diet if coarse and indigestible forms of starch are avoided; the predigested carbohydrate foods are to be preferred, especially in the more severe cases; while sugar is best given in the form of honey, malt extract or well-stewed fruits. Boas forbids cooked fruits, but probably this prohibition is mainly applicable to the more acid sorts.

All substances which are known to excite gastric secretion should be avoided, as increase in the total gastric juice will of course produce an increase in the absolute amount of hydrochloric acid, though not in the percentage. These substances are red meats and meat extracts, meat juice, salted meats, brown bread, yolk of egg, skim-milk, spices and condiments and all highly seasoned dishes. Effervescent drinks are excluded, as carbon dioxide is a gastric stimulant, and some authorities also exclude alcoholic drinks of all kinds. Possibly a little whisky or brandy well diluted with water may do no harm, but most patients will be better without them.

Boas finds that whereas coffee is always harmful tea is often well borne and cocoa generally harmless. The latter should always be made from the nibs, so as to include the fatty constituents. In this country, where tea is often taken strong and with little milk, and coffee comparatively dilute and with a considerable quantity of milk, I think that the latter is often found to be preferable.

An interesting point in connection with the diet in hyperchlorhydria is the effect of common salt. The normal average stomach secretes about 7 grm. of chlorine in the form of hydrochloric acid in twenty-four hours, whereas the total chlorine content of the blood has been calculated at only 11 grms. The hydrochloric acid secretion therefore is partly dependent on the amount of chlorine ingested as common salt. Some years ago Lanfer reported certain cases of hyperchlorhydria which, though somewhat severe in type, did well on an ordinary mixed diet without the addition of salt. If, however, they took 2 or 3 grm. (30 to 45 gr.) of salt with their food, an access of pain occurred. Bickel states that salt solutions over .9 per cent. are stimulants of gastric secretion, so that there appears to be a double reason for restricting the salt intake.

To pass on to the drug treatment, it is of course well known that aperients and alkalies are the main requirements of these patients. Mild cases often do well with an ordinary alkaline bismuth mixture an hour or two after meals. A glass of Vichy or Apollinaris water first thing in the morning is also very useful. Some authorities forbid the use of aperients, and rely on careful diet or abdominal massage, but I believe in most cases the ordinary saline aperients are quite well tolerated. Robin advises rather complicated alkaline mixtures in the more marked cases, such as the following:

℞ Magnesiæ	. . . .	20 gr.
Sod. Bicarb	. . . .	15 ,,
Pulv. Sacc. Alb.	. . .	30 ,,
Codeinæ	. . . .	$\frac{1}{8}$,,
Calcii Carb. Bism. Subnit.	āā . . .	12 ,,

This is given in the form of a powder stirred up in water, just before the pain is likely to begin. The sodium bicarbonate acts immediately and the others more slowly, and a large amount of CO_2 is not given off at once as is the case if large quantities of bicarbonate are given at a time. If there is diarrhœa, the calcium and bismuth salts

may be increased, and the magnesia diminished. Belladonna in the more severe cases is useful as a temporary measure. Robin's plan of giving it before meals combined with opium and hyoscyamus is probably the best, though some prefer to give it immediately after food.

Those who have a preference for the newer drugs may use *eumydrin* (atropine-methyl-nitrate), which has a similar action to atropine, in doses of $\frac{1}{30}$ to $\frac{1}{60}$ gr. in milk sugar immediately before meals. It has the advantage of a low toxicity, but has been found to diminish appetite.

Neutralon is aluminium silicate, and is broken up by the free HCl into silicic acid with the formation of aluminium chloride, an astringent and antiseptic according to Zweig. A teaspoonful of powdered neutralon can decompose 400 c.c. (about 14 oz.) of a ·2 per cent. solution of HCl at body temperature in one hour. A teaspoonful may be ordered suspended in a wineglassful of water quarter to half an hour before food. The dose may be repeated after food if necessary.

Peroxide of hydrogen is said to act by increasing the amount of mucus in the stomach, and thus protecting the mucous membrane from the acid gastric contents. It may be given in the form of $\frac{1}{2}$ to $\frac{3}{4}$ per cent. dilution of the ordinary H_2O_2 solution; the dose is 10 oz. before meals; or two $\frac{1}{2}$ grm. tablets of Merck's magnesium perhydrol may be administered in the same manner.

May 5th, 1913.

Ligneous Phlegmon of the Abdominal Wall.—W. W. Grant reports two cases of this condition due to staphylococcic infection. The disease generally occurs after middle life in persons whose resistance has been impaired by previous ill-health. The immediate cause is a slow infective process with or without trauma. The dictum of Reclus that it is not due to specific bacteria but to a variety of germs of mild virulence seems confirmed. Staphylococci seem to be the chief offenders. The usually slow and protracted course and final resolution are characteristic. Hyperleucocytosis always exists. The disease is cured more quickly and completely by free incision and serum treatment than by any other means.—*Med. Record*, vol. lxxxiii, p. 721.

SOME REFLECTIONS SUGGESTED BY A BLADDER CASE.*

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

MR. PRESIDENT AND GENTLEMEN,—The case I am bringing forward this afternoon is that of a fruiterer and greengrocer, æt. 55 years, of a highly neurotic temperament. Up to September 27th he was in his usual health, and had never had any genito-urinary symptoms. On that day, however, while lifting some heavy bunches of bananas he felt something "give" inside him, and immediately was seized with severe pain in the right abdomen, radiating towards the back, but not shooting either into the groin, testicle, or thigh. The pain was increased on movement, but disappeared on lying down. He rubbed the painful parts with camphorated oil. Four or five days later he passed a few drops of blood towards the end of micturition. After fourteen days the original pain disappeared, to be succeeded by pain referred to the tip of the penis, coming on towards the end of micturition and lasting for a minute or two. The frequency of micturition was also increased, the patient having to pass water as often as twenty to twenty-five times during the day and night. He described the water as being on one occasion like crusted port.

When I first saw him, at the beginning of December, ten weeks after the onset of his illness, the pain had become continuous, and was of a dull aching character, extending along the whole length of the penis, but most marked at the tip, and being greatly increased just before and after micturition, when he described it as resembling "the cutting of knives." He also complained bitterly of rectal tenesmus on micturition. There was no pain in the kidney region, though once I elicited some tenderness on pressure in the right renal angle. As before, the pain did not radiate into the groin or testicle. There was no discharge from the urethra; there had been no hæmaturia for fourteen days.

Subsequently the condition of the urine varied, at times being quite clear for a week or more, and then containing a deposit of blood and small clots,

* Read at a meeting of the Devon and Exeter Medico-Chirurgical Society.

and on several occasions a little clear blood was squeezed out of the bladder towards the end of micturition or during defæcation. On one occasion there was a copious deposit of large uric acid crystals and of oxalates. The frequency of micturition fell to about twelve times during the twenty-four hours, and was not increased at night.

On two occasions I sounded his bladder, with a negative result. On neither occasion, however, was the examination followed by any hæmorrhage. Examination *per rectum* revealed nothing abnormal, and posteriorly, at any rate, the prostate was not enlarged. The right kidney was not palpable.

Here was a case which commenced with the sudden onset of pain in the right abdomen, immediately after a straining effort. This was succeeded in a few days by the passage of some blood *per urethram*, and by increased frequency of micturition. Later the original pain disappeared to be replaced by pain in the penis, especially severe at the tip and aggravated by the passing of water. Hæmaturia continued intermittently. Physical examination yielded negative results. The history of a strain, followed by pain in the right abdomen, tending to spread round to the back, and the subsequent hæmaturia, naturally suggested some slight injury to the kidney. Whatever the cause of the primary pain, whether it was due merely to a muscular strain, or whether something had really gone wrong with the kidney, the subsequent course with the character and incidence of the pain, and the hæmorrhage coming on principally towards the end of micturition, pointed to the bladder as the seat of the trouble. Sounding had yielded a negative result, the prostate was not enlarged, at any rate towards the rectum, nor were there any other symptoms of prostatic disease. The general condition of the patient negatived any constitutional cause for the hæmaturia, such as scurvy, purpura hæmorrhagica, hæmophilia, etc. Putting aside such unusual causes as *Bilharzia hæmatobia*, the case was narrowed down to one of ulceration, simple or tubercular, or of new growth.

As regards simple ulceration, none of the causes of this, such as stone, chronic cystitis, etc., was present, and the age of the patient was against the case being one of what Mr. Fenwick calls "simple solitary ulcer," which he tells us occurs in young adults.

The age of the patient, again, as well as the amount of the hæmorrhage, which at times was considerable, the absence of pus from the urine, and no evidence of tuberculosis, either local or general, being discoverable, was against tubercular ulceration. Hence one was driven to the diagnosis of tumour of the bladder—a papillomatous or so-called villous growth, or a malignant tumour.

As regards villous tumours Mr. Southam says ('Encyclopædia Medica,' vol. i): "In the early stages attacks of *painless* hæmaturia, with intervals of complete freedom from bleeding, are usually the only symptoms present. . . . It will often be found that after sounding the bladder bleeding is excited or increased." On the other hand, he says: "In cases where the bleeding is irregular or more or less persistent, but *preceded or accompanied from the first by pain and increased frequency in micturition*, the growth is more likely to be cancerous, viz. epithelioma." (Italics my own.)

Taking all these points into consideration, one came to the conclusion that this patient was suffering from a tumour of the bladder, probably malignant. But all the evidence was merely circumstantial.

Considering a cystoscopic examination necessary, I took the patient to Mr. Freyer, who found "a large lobulated tumour with a very broad base growing from the upper aspect of the bladder right to the orifice of the urethra, which is undoubtedly malignant." He did not think the patient's condition would be at all improved by operation, and considered that whatever operation was undertaken the growth would recur. Thus, less than four months from the appearance of the first outward and visible sign, this growth had spread to such an extent that operation was out of the question.

He gradually sank, worn out by pain and hæmorrhage, and died on August 19th, less than eleven months from the onset of his first symptom.

The case presents some points of considerable interest. The symptom of terminal penile pain is generally regarded as indicative of stone in the bladder; no doubt this is the commonest cause, but other diseased conditions of the bladder, as in the present case, may also give rise to it; moreover, as pointed out by Sir Wm. Bennett in a lecture of his on this very symptom ('Clinical Journal,' October 14th, 1903), conditions entirely outside the bladder may also be responsible for it. He

narrates several cases in which the classical symptoms of vesical calculus were present, but in which the stone was found, not in the bladder, but in the lower part of the ureter, below the brim of the pelvis. Commenting on this he says: "You have, I doubt not, been told that the bladder as it becomes empty leads to the stone or other mass being forced into the neck of the bladder, thus giving rise to the reflex pain felt at the tip of the penis—indeed, it is sometimes said that unless the stone or other material is capable of being so caught in the neck of the bladder, the terminal penile pain cannot occur. If this is true it is clear that by no possibility could a stone in the ureter produce pain at the tip of the penis; but, as a matter of fact, it is not necessary for a stone or any other foreign body to be in actual contact with the neck of the bladder to produce pain at the tip of the penis, because there are other causes entirely outside the bladder which will produce that particular symptom." He adds: "It is useful to bear in mind this fact, to which I do not recollect having seen any reference (this was in 1903), because, supposing in a case in which the characteristic pain at the tip of the penis is present, you fail by all your methods of examination to elicit evidence of the existence of any stone, other foreign body or new growth, especially villous growth, in the bladder, you must look further afield for your explanation."

Now why should a stone or other foreign body in the bladder, or a vesical new growth, give rise to pain referred to the tip of the penis and the perinæum, and sometimes extending to other parts, such as the thigh? It is easy to dismiss the matter by saying that the pains are reflex in character. This is certainly true, but I think it will greatly assist us in correlating many apparently dissociated symptoms, and in making many of the crooked paths of diagnosis straight, and the rough places plain, if we consider the course of these reflex sensations.

I fear we sometimes forget that, were it not for the work of anatomists and physiologists, we should not, in a state of health, know anything of the size, shape, position or movements of our internal organs. Which of us, for instance, is conscious of the possession of an appendix, much less of its size, shape or direction? Again, take the mucous membrane lining the alimentary canal, which, as you will remember, from the lower part of the pharynx until we reach the anus is developed from the hypoblast. This, so far as the conscious self is concerned, is a *terra incognita*, not being endowed with ordinary sensation, nor with the power of localisation. The mouth and the pharynx at the one end and the anus at the other, however, possess these powers to a greater or less extent, and in this connection it is interesting to recall that the lining membrane of both these latter parts is formed by invaginations from the epiblast, the layer from which the great cerebro-spinal nervous system, the nervous system of the conscious self, is also developed. Now the mucous membrane lining the bladder and urethra is developed from the hypoblast, and hence we should expect that this also would be devoid of ordinary sensation and of the power of localisation, which it is. Thus a patient with a stone, or other foreign body in his bladder, or with a vesical new-growth does not come complaining that he has something in his bladder, but that he suffers from pain at the end of the penis when he passes water, the part where, as in the case of the anus, the skin derived from the epiblast joins the mucous membrane derived from the hypoblast. Whether or not the epiblastic layer is continued into the fossa navicularis I cannot ascertain, but it is a significant fact that the epithelial lining of the urethra is of the columnar variety excepting at the fossa navicularis, where it is laminated.

Dr. J. Mackenzie, in that charming and suggestive little book of his, 'Symptoms and their Interpretation,' proves, I think conclusively, that pain in visceral disease is felt, not in the organ itself, but in the distribution of one or more of the sensory cerebro-spinal nerves, which are in relation with the sympathetic nerves supplying the particular viscus in question, a point which has also been worked out very fully by Dr. Head.

The autonomic or sympathetic nervous supply of the parts at present under consideration is derived from the inferior hypogastric or pelvic plexuses, which send secondary plexuses to the rectum, bladder, prostate and penis, and in the female to the uterus and vagina. Now the sympathetic nervous supply of the fundus or base of the bladder is in relation, *viâ* the vesical and pelvic plexuses, with the lower part of the sacral plexus of the cerebro-spinal portion of the nervous system,

especially with the pudic nerve with its trinity of branches, distributed respectively, the hæmorrhoidal to the anus and surrounding parts, the perinæal to the perinæum and under part of the penis, and the dorsal nerve of the penis to the integument of the upper part of the penis as far forwards as the meatus.

Now, stimulation in the course of or at the origin of a sensory nerve gives rise, if strong enough, to pain referred to its peripheral distribution, so that a sufficient irritation in the region of the fundus of the bladder, by exciting the origin of the pudic nerve, will give rise to pain referred to the termination of one or more of its branches, and among other places to the tip of the penis, which might well be called the chief spokesman of the bladder.

But you may say: "How does this explain the presence of this particular symptom when a stone is situated, not in the bladder, but in the juxta-vesical portion of the ureter?" For clinical purposes I think it is best to regard the bladder as consisting of two segments, an upper and a lower, the lower being far more sensitive than the upper, which latter constitutes the body of the bladder, and reacts, like other hollow unstriped muscular organs, to distension rather than to direct irritation. Dr. Mackenzie, in the work already cited (p. 183), says: "The bladder in its development is derived from two sources, the allantois and the cloaca. From the former there comes the upper division, and from the latter the fundus of the bladder (the trigone). The nerve supply of the bladder is likewise from two sources, from the upper lumbar region, and from the sacral autonomic (second and third sacral). As the result of this nerve supply the sensory symptoms are felt in two regions, in the hypogastric region where the upper lumbar nerves are distributed, and in the perinæum and along the penis where the sacral nerves are distributed."

Now the lower part of the ureter opens into the base of the bladder, and developmentally, as well as by its nerve supply, which is derived from the vesical plexus, and also clinically as evidenced by the cases narrated by Sir William Bennett, belongs to the lower rather than to the upper segment of the bladder. Hence in juxta-vesical ureteric stone pain is felt at the tip of the penis during micturition owing either to the ureter itself contracting upon the stone, or to the pressure exercised upon it by the contracting bladder, for the ureter pierces the bladder-wall very obliquely, lying imbedded in its muscular tissue for nearly three quarters of an inch.

Similarly the rectum also may be regarded as consisting clinically, as it does developmentally, of two segments, an upper and a lower, the latter of which is, as in the case of the bladder, far more sensitive than the former. Mr. Bowlby, in a lecture on "Obstruction of the Large Intestine" (CLINICAL JOURNAL, July 21st, 1909) says, "When cancer occurs in the rectum it is often in its early and removable stages painless. In proportion as it is near to the anus it gives rise to abnormal sensations or to real pain. The nearer it begins to the anus the sooner will the anus be infected by invasion, and the earlier it is affected the earlier the patient appreciates the presence of something abnormal and seeks advice. When the cancer is three or four or five or six inches up it is liable to cause no pain whatever, and in many cases it is of considerable size, before the patient has any idea that there is anything serious the matter with him at all."

As an illustration of this I may mention the case of a gardener, only æt. 36 years, who consulted me in September, 1910. He was apparently in robust health, but complained that for some months his bowels had been troublesome, sometimes not acting for several days. He also thought that there was a lump inside his rectum. On several occasions he had passed some blood, but had suffered *no pain whatever*. On rectal examination I could discover nothing abnormal, and therefore treated him for constipation with what seemed to be a satisfactory result. He consulted me again, however, in the January of the following year, and this time, the former treatment not proving successful, I again made a rectal examination, and to my dismay discovered a hard, nodular growth just within reach of my finger, surrounding the rectum. I at once sent him to an operating surgeon, who reported the growth as inoperable, all he could do being to perform a colotomy, after which the patient lived for nearly two years.

I must confess that, seeing I could find no adequate cause for the hæmorrhage, I consider myself to blame for not having had the patient examined by the sigmoidoscope, for no doubt the growth would then have been discovered while it was still in an operable condition. It is not a com-

forting thought, gentlemen, to reflect that some of us learn only by bitter experience, gained not infrequently, it must be confessed, at the expense of our unfortunate patients.

These remarks of Mr. Bowlby concerning the rectum apply, I think, *mutatis mutandis*, to the bladder, and it seems to me that so long as the growth is confined to the upper segment of this viscus, the patient may suffer little or no pain, and it is only when the lower segment is invaded that pain becomes a prominent symptom. It is difficult to believe that in the case at present under consideration the growth had spread to such an extent as was revealed by the cystoscope, in less than four months—the time elapsing between the onset of the first symptom and the discovery of the growth.

And now a word or two as to so-called "pain in the bladder." To borrow the expression of a celebrated statesman, on a memorable occasion, it seems to me that there is a certain amount of "sloppiness" in our use of this term. As we have seen, the bladder does not possess the power of ordinary sensation nor of localisation. I grant you that in over-distension and some other morbid conditions of the bladder, the patient suffers from pain which he refers to the hypogastric region, and this, in conjunction with our knowledge of the position of the bladder, and with the presence of other symptoms pointing to this organ, such as retention or incontinence of urine, leads us to conclude that the patient has pain in his bladder. But an exactly similar pain is present in colic of the lower intestine. This point is well illustrated by the case of a gentleman to whom I was called early one morning, and who was suffering from severe pain in the abdomen and had also vomited. On my arrival at the house at 7.30 a.m., I found him lying in bed, but not with his knees drawn up. His pulse was only 54, very feeble and intermittent, his temperature 95·8° F.—that is to say, he was in a state of collapse. Soon after my arrival he was seized with a severe attack of pain and rolled about in bed groaning in agony. There was a history of some dietetic indiscretion. When asked where his pain was, he put his hand on the hypogastric region over the position of the bladder. When questioned closely, he told me that each attack of pain commenced in the lower part of the epigastric region in the middle line; it then worked down to the region of the umbilicus, and finally finished up in the hypogastrium—the typical distribution of the pain in cases of intestinal colic, as depicted in a diagram in Dr. Mackenzie's book (*op. cit.*, p. 113). There was an entire absence of urinary symptoms, and for this and other reasons I diagnosed intestinal colic, which the subsequent course of the case proved to be correct.

In speaking of "bladder pain" Dr. Mackenzie says (*op. cit.*, p. 183): "The pain from an over-distended bladder, as in retention of urine, is felt mainly above the pubes, and resembles in its situation and character the pain of uterine contraction, or *the pain of peristalsis of the lower end of the great gut.* This pain is intermittent in character, and is due to the passage of waves of contraction over the bladder."

Again it is interesting, in this connection, to recall that developmentally the body of the bladder is, in reality, merely a diverticulum from the lower part of the large intestine. You will remember that it is from the hind gut, at an early stage in the development of the embryo, that the allantois springs, and that it is the lower portion of this outgrowth which ultimately dilates to form the body of the bladder, the structure of which, with its peritoneal coat, its muscular coat, containing longitudinal and transverse fibres, its submucous and its mucous coats, is essentially the same as that of the large intestine. Hence it is not surprising that pain felt as the result of the violent contraction of both these organs should be referred to the same region.

I mentioned that this patient complained bitterly of rectal tenesmus during micturition. This is another example of the intimate relations between the bladder and the rectum. Not infrequently rectal symptoms are the result of some morbid condition of the bladder, and, on the other hand, symptoms pointing to the bladder may arise in connection with disease of the rectum. In the female the uterus and vagina, which also receive their nervous supply from the pelvic plexuses, intervene to further complicate matters.

A little boy was suffering from symptoms suggestive of stone in the bladder. On inquiring after the condition of the bowels, I was informed that they were very obstinate, and on examining the rectum for a possible polypus, I discovered it to be full of hard, dry, gritty fæces. The cure of

the constipation was followed by an entire disappearance of the bladder symptoms.

You will remember that the centres which preside over the organic reflexes of micturition and defæcation are situated close together in the lumbar cord, and, as Dr. Mackenzie points out (*op. cit.*, p. 84)—"A stimulus reaching the spinal cord may, if of adequate strength, stimulate other nerve-cells in its neighbourhood, and these respond *according to the nature of their function.*" (italics mine). Hence one can understand how a strong stimulus from the bladder may, so to speak, overflow the micturition centre and flood that for defæcation, with the result that the patient suffers from rectal tenesmus, which might well be called "dry defæcation," analogous to the dry cough which takes place when the coughing centre is stimulated but there is no *materies morbi* to be expectorated. On the other hand, excessive stimulation of the defæcation centre may, in like manner, give rise to distressing vesical tenesmus.

In this connection the following passage from the lecture by Sir William Bennett, already referred to, is of considerable interest: "I have seen three cases in which the predominant symptom in fissure of the anus was pain at the end of the penis, exactly like that produced by stone in the bladder. In point of fact, in two cases the patients consulted surgeons because the possibility of bladder trouble had been suggested, some little discomfort that had occasionally been felt in the rectum having been overlooked. The cure of the fissure in each case led to the disappearance of the pain at the tip of the penis."

These cases illustrate not only the fact that rectal disease may give rise to symptoms suggestive of bladder trouble, but also emphasise the point, already referred to—that pain at the end of the penis may be the result of causes entirely outside the bladder. In these cases, however, I should think the pain ought to be regarded as a referred rather than a reflected pain, seeing that both the anus and the tip of the penis are supplied by different branches of the same cerebro-spinal nerve—the pudic—and we know that irritation of one branch of a nerve may give rise to pain referred to the distribution of another branch of the same nerve—a fact which was painfully impressed upon me some time ago, when I suffered from severe pain limited to the lower jaw due to a decayed tooth in the upper jaw, the extraction of which afforded me complete relief.

It seems to me most unfortunate—I had almost said "impious,"—that two organs, the bladder and rectum, which Nature hath so intimately joined together, not only developmentally, but also structurally, physiologically, *and* clinically, should be so ruthlessly torn asunder by the hand of the specialist. There is, however, so much to be learnt nowadays, that I suppose division of labour, or, if you prefer it, devolution, is an absolute necessity.

May 5th, 1913.

Auto-Serotherapy in Sero-Fibrinous Pleurisy.—M. Fishberg believes that this method of treatment is a simple one, and ought to be tried in every case of pleural effusion in which the fluid is not purulent. Indeed, inasmuch as in practically every case of pleurisy showing signs of effusion exploratory puncture is to be performed with the view to determining the character of the fluid in the pleural cavity, it is advisable, when the contents of the pleural cavity are found to be serous, that the needle should not be withdrawn completely, but that when its point is brought to the subcutaneous tissue it should be injected then and there. The quantity of fluid to be injected subcutaneously need not be very large; from 2 to 5 c.c. is quite enough. In favourable cases it is observed that within two or three days the subjective symptoms—the pain, the dyspnœa, the prostration, etc.—are ameliorated or entirely disappear, while physical exploration of the chest shows that the upper level of the fluid is lower than it was before the injection. Within a week or ten days the fluid in the chest disappears altogether in about 80 per cent. of the cases. In some patients it may be necessary to repeat the treatment several times every two or three days before any salutary effects are evident. In about 10 or 15 per cent. of cases this mode of treatment seems to fail, but even in this instance no harm is done to the patient.—*Journ. A.M.A.*, vol. lx.

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A CLINICAL LECTURE ON PELVIC CELLULITIS.

By JOHN B. HELLIER, M.D.Lond.,

Professor of Obstetrics in the University of Leeds; Hon. Obstetric Physician to the Leeds Infirmary; Consulting Physician to the Leeds Maternity Hospital.

THIS is a short clinical study of 54 cases of puerperal pelvic cellulitis, otherwise called parametritis and pelvic phlegmon, treated by me during the last thirteen years in the Leeds Infirmary. For a full account of pelvic cellulitis the reader is referred to any good text-book. My present purpose is to sketch the most interesting features in this series. We have no proper lying-in beds in the Leeds Infirmary, but from a very wide area obstetric cases are sent in from time to time and are treated in the gynæcological wards. Perhaps an obstetrician who records such a series of cases of puerperal infection may be excused if he hastens to explain that he has had no responsibility for their production. No general practitioner is likely to have much personal experience of this condition in these days of aseptic midwifery and this record may interest many. At the same time it may be affirmed that even the most careful man will see occasional examples of the disease, for the environment of parturient women is rarely entirely within our control except in the case of those who enter hospital or nursing home some time before labour begins.

Cases of puerperal infection are roughly divisible into three groups. The first includes putrescence of the lochia or of retained secundines causing sapræmia, the germs not spreading beyond the uterine cavity. Most of these yield to irrigation with perhaps the use of the blunt flushing curette, and the patients rise from childbed without much prolongation of their lying-in period and are said

to be cured, although some of these reappear later at the gynæcological clinic, suffering from chronic endometritis, etc. Some run into the next class, but on the whole the prognosis is good. A large proportion of the cases of slight "morbidity" in childbirth come under this head.

The second class includes cases where there is acute recent infection which does spread into the uterine wall and beyond the uterus, and this class embraces the most serious and fatal forms of puerperal infection. The infection is often very virulent, and the lesions are diffuse and multiple. In cases examined post-mortem, the lesions most commonly found are suppurative peritonitis, purulent cellulitis, septic metro-phlebitis with septic thrombi in the pelvic veins and septic pneumonia. They run a rapid course and are little amenable to surgical treatment.

In the third class we place the more chronic infections. The lesions are later in onset and slower in development, and tend to be plastic and localised, and either recover spontaneously or lend themselves to surgical treatment. Amongst such conditions may be mentioned pelvic phlegmon, phlegmasia dolens, pyosalpinx, and tubo-ovarian abscess and localised peritoneal abscess. It is the first of these that is selected for study here.

Pelvic phlegmon or cellulitis is a very striking and interesting condition, and it is characterised by the formation of a localised pelvic or pelvi-abdominal swelling due to inflammatory exudate, which often becomes exceedingly hard, so that it has received the name of "plaster-of-Paris" exudate. To-day we see little cellulitis other than puerperal, but many years ago, before gynæcological procedures were asepticised, the non-puerperal form was more common. It used to follow the use of the old-fashioned "hysterotomes" and "metrotomes," now seen only in museums, and it was often caused by the free application of potassa fusa to the cervix for "areolar hyperplasia." I once had the opportunity of watching such practice for a year, and have vivid memories of hard exudates and especially of the curious "horse-shoe" exudate, half around the cervix. I have not seen this for very many years but it is depicted in the older text-books. See, for example, a sketch by Dr. Robert Barnes in his 'Diseases of Women,' 1878, p. 583.

Apart from labour, operative interference or trauma a diagnosis of simple pelvic cellulitis is rarely accurate, some intra-peritoneal lesion being more likely to be present.

There is an acute form of cellulitis which is but part of a diffused acute infection and our series affords two illustrations.

Case 1.—Mrs. E—, æt. 35 years, had a miscarriage August 10th, 1910, being attended by a midwife, and was admitted August 12th in a desperate condition. She was treated by saline injections and anti-streptococcic serum but opera-

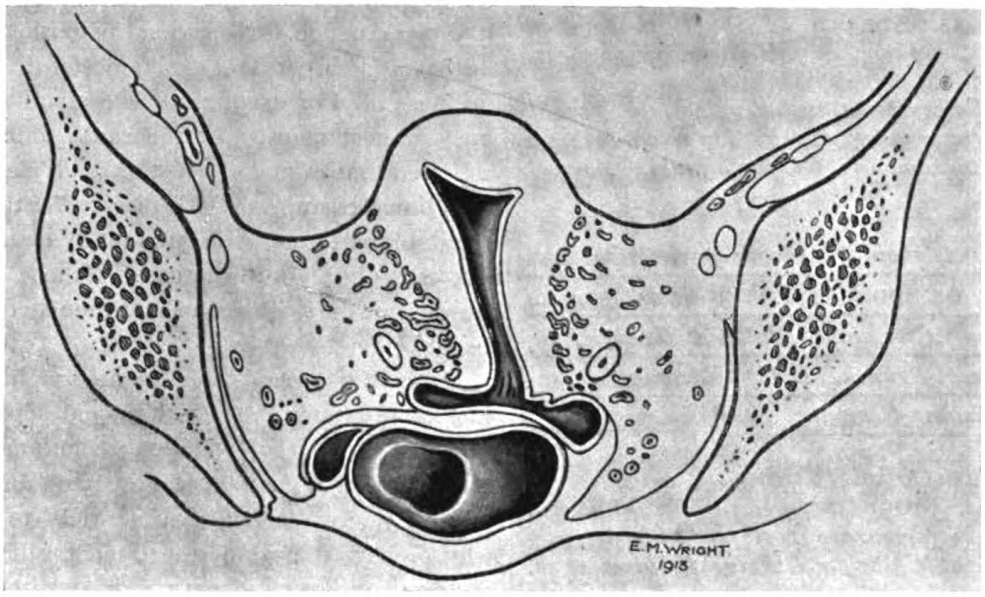

Fig. 1.—Section of pelvis in line of attachment of broad ligament. The vessels in the ligaments are shown.

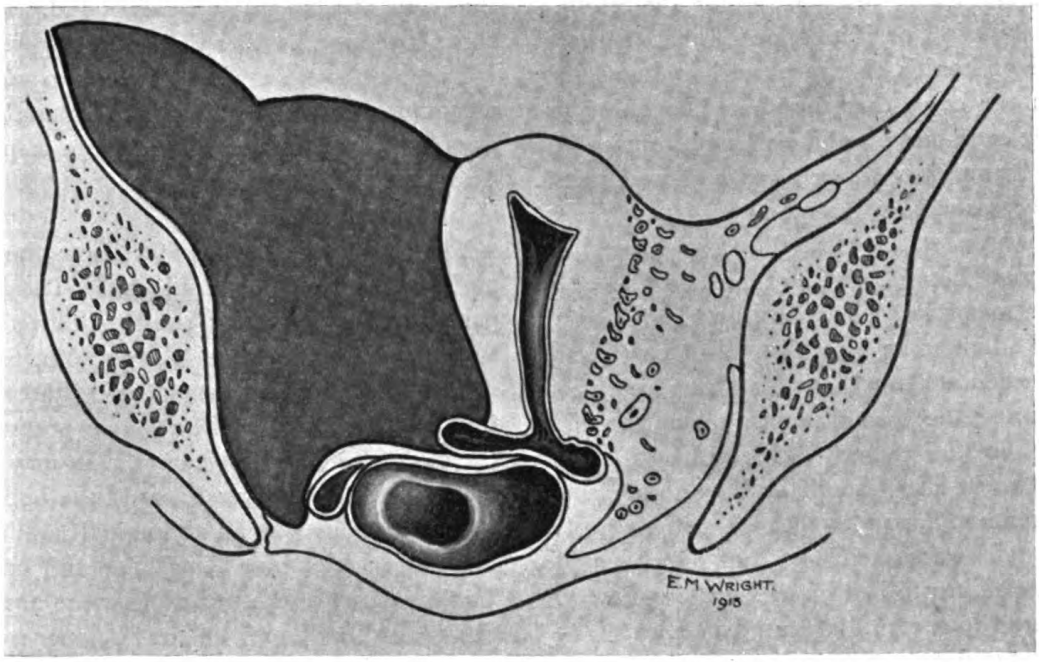

FIG. 2.—Shows diagrammatically a phlegmon in the broad ligament extending into the iliac fossa.

tive treatment was out of the question, and she died in twenty-four hours. On autopsy a piece of placenta was found in the uterus, the broad ligaments were infiltrated with pus which exuded from between their layers, and there was purulent fluid in the peritoneal cavity with flakes of lymph on the intestines. Cultures of streptococcus were obtained from the pus. The association of cellulitis with peritonitis in these acute suppurative cases is very characteristic.

CASE 2.—Mrs. B—, æt. 34 years, was confined outside the infirmary on December 26th, 1904. On the second day she had a rigor and early became jaundiced. The uterus was explored with curette, and the posterior *cul-de-sac* opened, with negative result. Died twelfth day after delivery. Post mortem a collection of pus and blood was found in the right broad ligament, the ovarian vein was thrombosed and there was septic bronchitis. The infection here was highly virulent.

The remaining cases were all of the chronic kind. In every case there was an inflammatory swelling in the cellular tissue of the pelvis. Twenty-five underwent spontaneous resolution and twenty-seven suppurated. In every case, except in specified exceptions, the abscess when opened was entirely extra-peritoneal.

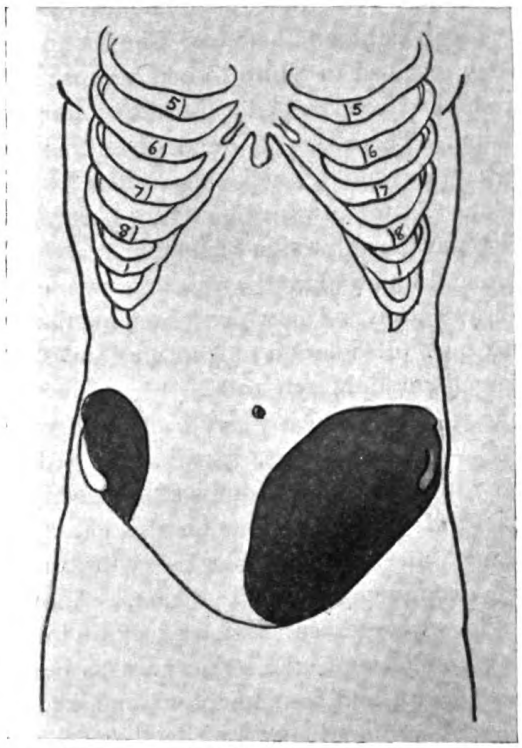

FIG. 3.—Shows on the left side a very large swelling above the groin reaching further towards the navel than usual (Case 14). On the other side is seen the position of a " remote " phlegmon (Case 6).

In the cases that resolved, either the cellulitis was the only lesion, or, if it happened that there was also some peritonitis, that recovered spontaneously also. In the cases that suppurated, opening the abscess by extra-peritoneal incision was followed by cure of all symptoms except when otherwise stated. All these cases were of puerperal origin.

I have been struck with the long periods that have elapsed between labour and the time when admission to hospital has been sought. I find that four were admitted in less than one month after delivery, seven at about the end of the month, about twenty during the second month, ten in the third, one each in the fourth, fifth, sixth and eighth months, two in the seventh and two in the ninth. A very common history is that the patient got up in about ten to fourteen days, but after a day or two had to return to bed on account of pain.

Here are two cases of late admission.

The first is also interesting because it was one of the few cases in which the extra-peritoneal seat of the abscess was verified by abdominal exploratory incision.

Case 3.—Margaret M—, æt. 31 years, was admitted January 15th, 1910, eight months after the delivery of an eight months fœtus. She got up on the tenth day, but had to return to bed because of the pain; she had been in bed most of the time up to admission. There was a rounded swelling above the left groin and a thickening in the left broad ligament. As there was some doubt as to whether cellulitis was the only lesion I explored by abdominal section, and found that the swelling was entirely intra-ligamentous and extra-peritoneal and that there was no pus. Resolution took place, and she was discharged cured March 10th.

Case 4.—*An example of suppurating phlegmon.*—Mrs. C—, æt. 40 years, 7-para, was admitted March 14th, 1912, nine months after delivery. On vaginal and rectal examination the left anterior quadrant of the pelvis was blocked with exudate as shown diagrammatically in Fig. 4. Above the left groin was a crescentic swelling shown in Fig. 5, and it extended above the pubic crest as indicated in the diagram and fluctuation was marked. On March 15th an incision was made above the groin extra-peritoneally and the finger passed into an abscess which extended down behind the pubic ramus.

The abscess granulated from the bottom and she was discharged on May 2nd, about ten and a half months after delivery.

These cases are typical of the two varieties of phlegmon, non-suppurating and suppurating, and it does not seem necessary to quote many others. Those that follow have for the most part some special feature of interest.

The accompanying sketches show the common positions occupied by the phlegmon. The abdominal part forms a crescentic swelling above Poupart's ligament on one side (Fig. 2) or both, usually one side only, but perhaps extending behind and above the pubic symphysis. The pelvic portion usually occupies one broad ligament. It may be large, pushing the uterus over towards the opposite side, and continuous with an abdominal swelling above one groin; or it may be smaller and confined to the lower part of the broad ligament and close to the uterus, or it may lie in the upper part and be more "remote" from the uterus (Fig. 3). Usually the uterus is fixed, but sometimes it preserves some mobility. The great hardness often noticed in these swellings has been already mentioned. When the phlegmon suppurates the swelling softens in some part and may show signs of pointing, for instance. The tissues above the groin may become more or less boggy and the skin may redden. Sometimes a small focus of suppuration is found surrounded by much induration, which clears up after incision of the focus.

Here is a typical case, many similar being included in the present series.

Case 5.—K. S— was admitted May 28th, 1908, æt. 19 years, primipara. She had been delivered by forceps seven weeks previously, and had a rigor a few days later. On admission the uterus was fixed with pelvic induration all round it, especially on the left side, and there was a swelling above the groin over which the skin was red and tender. An incision made there allowed one to pass the finger into a purely extra-peritoneal abscess. This extended upwards towards the iliac fossa and not much into the pelvis. About five ounces of fœtid pus were obtained, and *Bacillus coli* and streptococci were found in it.

The patient made a good recovery and was discharged cured.

Case 6 is an example of "remote" phlegmon, and is also interesting because the nature of the swelling was determined microscopically. Mrs.

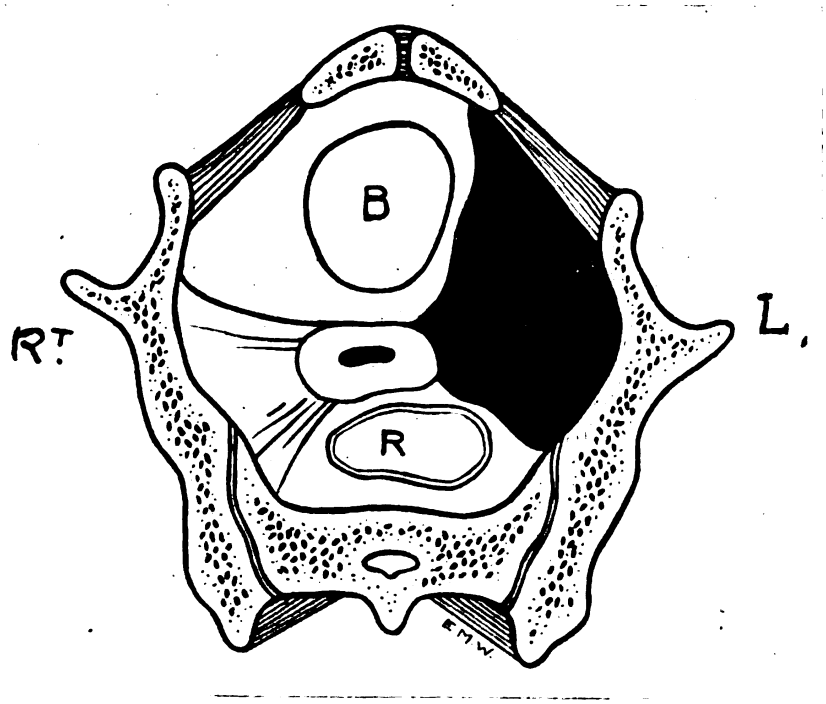

FIG. 4.—Refers to Case 4, and shows a horizontal section of a pelvis with phlegmon as felt by vagina and rectum.

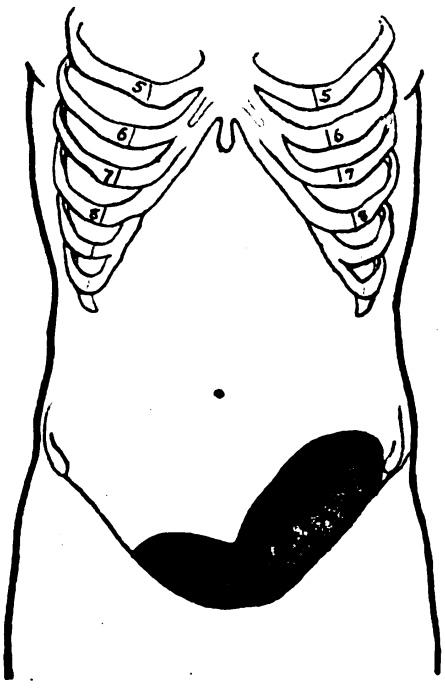

FIG. 5.—Shows the exudate in Case 4.

P—, æt. 29 years, 6-para, was admitted April 15th, 1909, eight weeks after instrumental delivery. On admission there was a semicircular swelling above the right groin with very little pelvic swelling. As it was uncertain whether this did or did not contain pus, and as the temperature was suggestive of this, an exploratory extra-peritoneal incision was made above the groin. It is doubtful if a drop of pus was obtained. The swelling consisted of hard brawny tissue and a small portion was examined by the pathologist. It showed chronic inflammatory changes, the cells being markedly polymorphous with a great deal of fluid exudate between them. She made a good recovery.

In only three cases the swelling lay principally in front of the uterus, between it and the bladder. One has points of interest that lead me to quote it.

CASE 7.—I was asked by one of my colleagues to see a girl who had a fluctuating swelling just above the pubes. She was not married. I opened an abscess and evacuated a considerable amount of pus. Knowing how rare such an abscess is apart from puerperal conditions and learning that there had been some uterine hæmorrhage, I

curetted the uterus and obtained some tissue in which chorionic villi could be demonstrated. Hence one could attribute the condition to a recent septic abortion which had probably been procured illegally, but I had no further proof of this. She recovered well.

One case in the series at least opened into the rectum, but it is possible that a small discharge into the bowel might escape notice; another certainly communicated with the bladder but required incisions elsewhere (see Case 12). In three other cases spontaneous discharge occurred; one opened above the groin (Case 8), and two into the vagina (Cases 9 and 10).

CASE 8.—M. H— was confined February 23rd, 1905. Clinical history defective, but had symptoms of infection soon after labour and secondary post-partum hæmorrhage in the third week. She was admitted July 24th, five months post-partum, and was in a very bad state with a bed-sore and with a sinus above the right groin discharging pus. It seems that the inguinal swelling had been mistaken for a hernia and *treated with a truss*. An extra-peritoneal incision allowed the finger to pass into a large cavity extending behind the pubic ramus. The uterus was but little fixed. Recovery was slow but complete.

CASE 9.—Mrs. T—, 1-para, æt. 30 years, was admitted ten weeks after labour with an offensive vaginal discharge, and was found to have a swelling in the left broad ligament discharging pus into the vagina through a small sinus. This was opened up with the finger and packed and irrigated and healed in two weeks.

CASE 10.—Mrs. R—, æt. 19 years, 1-para, admitted June 7th, 1905, ten weeks after an instrumental labour in which the cervix was lacerated; she is said to have had both primary and secondary post-partum hæmorrhage, and to have had rigors after labour. On admission there was free purulent vaginal discharge proceeding from a sinus in front of the cervix, and a finger passed into the utero-vesical space. Satisfactory recovery.

From time to time one sees a cellulitic abscess extending through the great sciatic notch, and one such occurred in this series.

CASE 11.—Mrs. W—, 5-para, was admitted November 6th, 1901, six weeks after labour. She had noticed a lump in the right buttock two weeks post-partum with tenderness. When admitted the temperature was 104° F. A soft fluctuating swelling was felt on the right posterior aspect of the uterus, which was not much fixed. Fluctuation could be obtained through from the buttock to this intra-pelvic swelling. External incision with suitable drainage was followed by cure in about six weeks.

I never saw a cellulitic abscess which burst into the general peritoneal cavity.

I have only once seen extensive burrowing from a cellulitic abscess under Poupart's ligament down into the thigh. The case, not one of the present series, came under my care in 1894 in the Hospital for Women in Leeds, and I quote it because it is one of the worst cases I ever saw with recovery.

CASE 12.—E. J—, æt. 29 years, 5-para, was admitted into the hospital April 9th, 1894. She had been confined at term in the previous February in very insanitary surroundings. She had a rigor on the third day, and rose from childbed in three weeks; but a fortnight later had to return to bed because of pain in the abdomen and in the left leg, and there was a swelling in left groin. *Per vaginam* the left lateral fornix was obliterated by a swelling which occupied the left side of the pelvis and extended above the groin and backwards towards the great sciatic notch. There was a temperature each night of 100° to 102° F. Aspiration of the swelling yielded no pus but some pus appeared in the urine, and the thigh and leg increased in size. On May 21st an incision was made above Poupart's ligament, and a large extra-peritoneal abscess was found extending along psoas and iliacus, and into pelvis and slightly into thigh. She usually kept her thigh drawn up, but extended it when anæsthetised. On May 30th there was much extension of abscess in the thigh, and incisions were required on the outer side of the thigh and on the inner side near the knee. The whole cavity was irrigated daily for several weeks with izal. The patient was reduced to a skeleton and was in a pitiable state, but ultimately the swellings all went down, every sinus healed, and she was discharged cured on July 17th, and two months later was found in excellent condition, walking well with the help of a stick. This was not a psoas abscess but an extension from a puerperal suppurating phlegmon (see Fig. 6).

The excellent recoveries obtained by simple extra-peritoneal incision are the rule in these cases, but it must not be forgotten that intra-peritoneal lesions may be present also, and if the simple

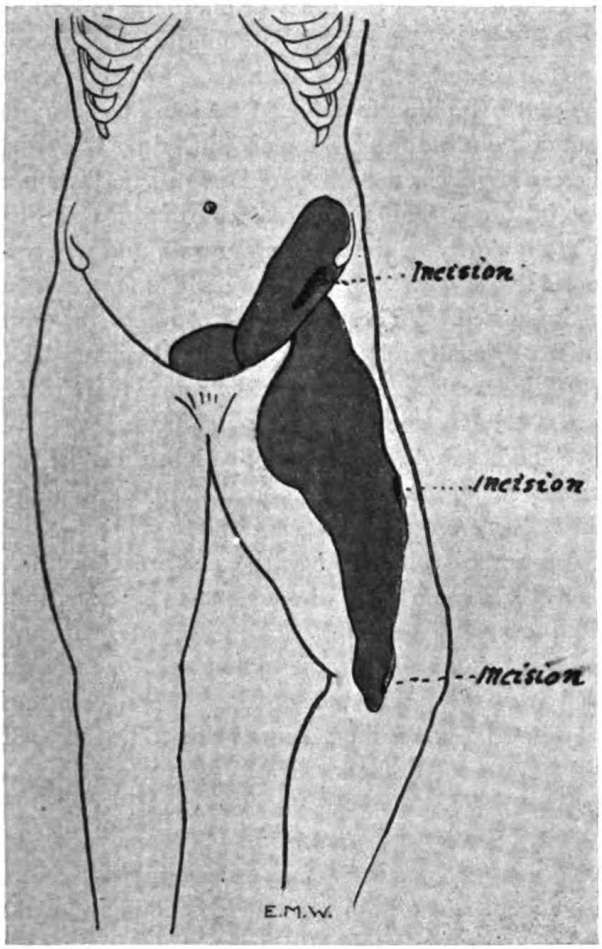

FIG. 6.—Illustrates Case 12, a suppurating phlegmon with extension into the thigh.*

extra-peritoneal incision fails to bring down the temperature and reach the focus of suppuration, then the abdomen must be explored. Here is a case in point where extra-peritoneal incision would have done no good.

CASE 13.—E. L—, æt. 29 years, 4-para, was admitted December 31st, 1911, one month after labour, having been in bed ever since with symptoms of pelvic infection. From admission till January 27th she was treated expectantly, the temperature oscillating between 99° and 102° F. She looked fairly well, and said that she felt well, and at first local signs were obscure. Then there developed a swelling gradually on each side of the uterus palpable *per vaginam*, and a swelling could be felt above each groin, but I could not be sure how far it was intra- and how far extra-peritoneal. On January 27th I explored by abdominal section. There was an abscess in the omentum, double tubo-ovarian abscess, and cellulitis in both broad ligaments. The omental abscess was very offensive. I removed part of omentum, corpus uteri, and appendages, and the appendix, draining the abdomen. Recovery was slow and complicated by obstruction of bowels, requiring re-opening of abdomen, but she made a very good recovery after that. The case shows the use of abdominal exploration in certain prolonged cases of puerperal infection.

Only one of these more chronic cases died, and that by an accident.

CASE 14.—Mrs. E— was admitted February 4th, 1908, about six weeks after delivery, complicated by adherent placenta, and she had a typical abscess in the left broad ligament, which I opened above the groin on February 17th. She did well at first, but the wound needed opening up on for better drainage, and she died suddenly under ethyl chloride, which was administered as anæsthetic. In this case the mitral valve was incompetent and there were pericardia adhesions.

Pathology.—Lymphangitis beginning between the layers of the broad ligament, and arising from an infected wound about the cervix, accounts for most of the cases. Fig. 1 shows a sketch of an oblique section through the pelvis in the line of the attachment of the broad ligaments to the sides of the uterus, and makes it easy to realise the genesis and relations of such exudates. Their course has been illustrated by the injection of plaster-of-Paris between the layers of these ligaments and watching its spread to the pelvic and abdominal wall, pushing up the parietal peritoneum in the process. The course of infections in the pelvis may be compared to similar conditions about the throat. Scarlatinal pharyngitis, for instance, extends by continuity of mucous surface along the Eustachian tubes to the tympanum, and also to the nose and adjoining sinuses, but it also spreads through the lymphatics, and we see the neck swollen level with the cheek, and lymphangitis may reach the deeper parts behind the pharynx. Likewise in the pelvis we see extension of infection along Fallopian tubes to peritoneum on the one hand, and through lymphatics to the cellular tissue on the other. At the same time the development of large phlegmons followed by com-

* I am indebted to Miss Ethel Wright for these drawings.

plete resolution is not seen in other parts of the body to anything like the same extent.

Symptoms.—The symptoms are those of local pelvic inflammation. A rigor may be the first sign of something going wrong, but this does not tell you what the lesion is. The first local sign, if you get the chance of investigating at the early stage, is often pulsation felt in the lateral *cul-de-sac*, and also local tenderness. Pain is often the first thing complained of, and it strikes through one hip. It is worse on walking, and the patient may keep one thigh partially flexed. The development of lateral swelling in one broad ligament is the first definite diagnostic sign.

Well-marked leucocytosis often marks the onset of suppuration. The temperature often runs very high, but I have been struck to see how well some of these patients bear a high rise of temperature night after night. They often look fairly well, and smile and say they have but little to complain of, except tenderness on movement or investigation.

Diagnosis. — There are two conditions that closely simulate pelvic phlegmon. The first is hæmatoma of the broad ligament. Here, too, we see the layers of the broad ligament separated by effusion, which tends to plastic hardening; it often forms a crescentic swelling above the groin, which the figures here given might well represent, and on rectal or vaginal examination very similar conditions are felt to those shown in Figs. 2 and 4. But its onset is apyrexial and more sudden; there is often a history of menstrual irregularity and signs of early pregnancy; moreover, the onset of the swelling is often accompanied by some vaginal hæmorrhage. A hæmatoma that is being absorbed and a phlegmon that is resolving may, apart from clinical history, be indistinguishable, and when a hæmatoma suppurates it is practically identical with a cellulitic abscess. But suppurating phlegmon is far more common than suppurating hæmatoma.

The other condition to be diagnosed from pelvic cellulitis is pelvic peritonitis with matted appendages, often thickened or cystic and with intestinal adhesions. Forty years ago, when obstetric physicians did not open the abdomen to remove diseased appendages, a good deal was called cellulitis that really was intra-peritoneal, and since then the importance of discriminating between the two conditions has been emphasised. I have not found much difficulty in the cases of puerperal phlegmon in this series. The pelvic swelling in phlegmon is unilateral, wedge-shaped, with base to pelvic wall; the pelvic swelling in adnexal tumours has a more rounded outline and is often obviously partly cystic, is often largely in the pouch of Douglas, and is often also bilateral. The abdominal swelling in cellulitis is really partly in the abdominal wall, and its crescentic shape, with base at Poupart's ligament, can be distinguished from a swelling felt *through* the wall and not specially related to Poupart's ligament. The cellulitic swelling rarely reaches more than three fingers breadth above the Poupart's ligament.

A good many slight cases of cellulitis occur, the exudate being limited and soon absorbed, and many such cases are overlooked.

Prognosis.—The early acute cases are very fatal.

In the chronic cases the prognosis is very good. Most of them get well and leave little if any permanent damage to the pelvic viscera, and do not interfere with subsequent utero-gestation. Pelvic peritonitis is much more likely to be followed by acquired dysmenorrhœa, pelvic pain and sterility.

Prophylaxis and treatment.—As one watches such cases running their course through many weeks one cannot resist pointing out to students that all the mischief has probably arisen from someone's transgression of the principles of aseptic midwifery, and that very many of the cases have a history of some manual or instrumental intervention in the conduct of labour.

This is not the place for a disquisition to students, but one may pause a moment to note what seem to be weak points in the technique of some men who have not been drilled in strict hospital routine. We cannot afford to do any operation without sterilised gloves. I always use them in midwifery practice. Their use is not universal amongst practitioners and is the exception with midwives.

There is very much to be said for always carrying a portable steriliser in the midwifery bag. The plan of carrying instruments in towels, etc., after sterilisation at home is very unsatisfactory if tested by proof cultures. But even if the forceps are brought sterile a blade may fall or be fouled, or the practitioner may have to hurry from one case to another. If the steriliser is ready at every labour, then forceps, scissors, gloves, douche tube, navel

ligatures, perinæum needle, etc., can be sterilised and resterilised at pleasure. I do not think it is easy to maintain asepsis through a long labour without great vigilance. Any man who carefully studies the prevention of puerperal morbidity will certainly increase his aseptic precautions and reduce intervention to the minimum.

The following point is worthy of note. It is never a light matter to pass hand or instrument from the lower part of the parturient canal to the upper. The upper part down to the os internum or a little lower is under ordinary conditions germ-free. The lower part always contains at least saprophytes. To smear the hand with vaginal mucus and pass it up into the uterus carries the germs up also. Many vaginæ contain other germs also, especially in multiparæ with lax vaginæ and prolapsing walls and with purulent leucorrhœas. Before gynæcological vaginal operations the most rigid disinfection of the vagina is carried out. The systematic irrigation of the vagina before every labour is not advocated, but before intra-uterine manipulations some disinfection is indicated. To cleanse with a swab in a holder dipped in soap spirit and then to douche with lysol is suggested. I was led to realise the difficulty of avoiding the conveyance of infection from vagina to uterus by observing cases when premature labour had been induced by passage of thoroughly sterilised solid bougies into the uterine cavity, and by finding that they were not perfectly sweet when expelled forty-eight hours or more latter. If they have caught up any infected mucus from vagina or cervix this is fully accounted for. Few things require more scrupulous care than to perform this minor operation with perfect asepsis.

It is a very wrong assumption to suppose that the accoucheur has the safety of the patient entirely in his own hands, and one on which the public need some enlightenment. If the husband sleeps by the patient's side till the first labour pain occurs the possible consequences must be borne in mind, especially if he be a man of uncleanly person. The uncleanliness of the patient herself and of her bed and clothing are very obvious in houses where nothing is clean at all, and the vagaries of nurses when beyond the doctor's observation are fraught with unknown possibilities. Till the old order of midwives have died out we shall never be free from unexplained infection in labour.

The excellent results obtained in this series are not due to any novelty in treatment. Almost all the cases have been dealt with by fomentations, hot vaginal douches, ichthyol and glycerine tampons and obvious symptomatic treatment until the temperature has subsided and resolution is progressing or till abscess has formed. Almost all the abscesses have been dealt with by incision above the groin, and they have drained and healed well, care being taken to prevent superficial healing before the deep part has closed. Only obvious vaginal pointing has led to vaginal incision. I am not fond of trying to reach the broad ligament by lateral vaginal incision. At the same time I am always ready to open the posterior *cul-de-sac* if there is any suspicion of septic fluid in the pouch of Douglas. Posterior colpotomy is the simplest and easiest and safest of all methods of opening the peritoneum but it is for peritonitis that it is useful and for localised peritoneal abscess. At one time I used to search for pus with the aspirator needle plunged into the exudate, but I have given this up because a negative result is so fallacious. Thick pus will not flow, and a small focus of suppuration surrounded by much plastic exudate is easily overlooked. If I explore at all I usually employ the knife, sinus forceps and finger. The deep epigastric artery runs from the point where the main artery is felt pulsating at the groin upwards and somewhat inwards and is easily avoided.

Serums and vaccines may help in the acuter stage and sometimes in the more chronic, but I do not use antistreptococcic or other serums when there is an unopened abscess. I usually give syrup ferri iodidi to help resolution, but have no experience of pelvic massage according to Brandt's method. I believe that the Bier plan of encasing the pelvis is a special appliance, and applying hot air has not been found very advantageous.

The care of the bowels needs no emphasising. Should there be any colitis, rectal irrigation is indicated.

May 12th, 1913.

A CLINICAL LECTURE

ON

CONDITIONS SIMULATING APPENDICITIS.

By HERBERT W. CARSON, F.R.C.S.,Eng.,
Surgeon, Prince of Wales's General Hospital, London, N.

THE differential diagnosis of appendicitis still presents many difficulties, and this is probably due to the fact that, in spite of the advances made in our knowledge of the condition, there are no exact methods of proving its existence. In many diseases microscopical and bacteriological research and the progress in X-ray diagnosis has allowed us to give a definite opinion uninfluenced by the personal factor, but for a diagnosis of appendicitis we must still depend upon trained observation, minutely accurate examination, and sound deduction from the observed facts. It is this personal factor which causes differences of opinion not only on questions of prognosis, but even of diagnosis. So much variation is seen in the symptoms and signs of appendicitis that it is perhaps permissible to say—and it sounds like a truism—that given average powers of diagnosis, the surgeon who sees the most cases makes the fewest mistakes. One great difficulty lies in the variable position of the appendix, so that an acutely inflamed appendix in the 11 o'clock position, *i. e.* pointing upwards behind the cæcum, will give rise to absolutely different signs from those of an acutely inflamed appendix in the 5 o'clock position, *i. e.* pointing downwards and inwards over the brim of the pelvis. Again, an acutely inflamed appendix pointing upwards gives rise to signs and symptoms referable to an area in the right hypochondrium (which is common to the gall-bladder, duodenum, pyloric end of stomach and head of the pancreas) or the right lumbar region, where disorders of the kidney must be differentiated, while if it points downwards a differential diagnosis must be made from diseases of the right appendages or even of the bladder. But perhaps the greatest difficulty exists in cases of chronic inflammation, for in this condition it frequently happens that we have to rely upon pain or tenderness (subjective signs in which there is a large margin of error), or even in some cases on the history of previous attacks. We know, too, that appendicitis occasionally simulates other diseases, the commonest of which perhaps is gastric or duodenal ulcer, and, indeed, we are beginning to realise that a pathological condition of the appendix may co-exist with, and even perhaps give rise to, disease of the upper digestive tract. So that in the absence of definite local signs—and they are often absent in chronic conditions—a diagnosis of appendicitis must be made with the greatest caution.

Conditions simulating appendicitis may be divided into two classes, according as they give rise to acute or chronic symptoms, remembering of course that the two classes overlap to some extent. Of the two, mistakes are much less serious in acute than in chronic conditions, for, as it is generally accepted now that acute abdominal disease should be promptly submitted to operation, the patient is no worse off if on opening the abdomen the supposed appendicitis turns out to be a ruptured pyosalpinx, or a perforated duodenal ulcer. But in chronic cases the matter is much more serious. We can all remember cases in which no improvement has followed appendicectomy undertaken for the relief of abdominal pain. It has been supposed that the continuance or recurrence of pain was due to adhesions resulting from the operation, and an opinion was given that matters would soon improve, but as we know that adhesions should not follow a well-conducted operation of appendicectomy in the quiet stage, the appendix, whether diseased or not, was probably not the real cause of the trouble. But we have, I hope, passed the period when it was considered sufficient, in removing a quiescent appendix, to withdraw the appendix from the abdomen through the smallest possible incision, and we realise now the necessity of examining the lower end of the ileum and the cæcum and ascending colon for adhesions in all cases where the appendix is to be removed.

CONDITIONS SIMULATING ACUTE APPENDICITIS.

The differential diagnosis of acute conditions which simulate appendicitis may be extremely difficult. The chief enemy to a satisfactory examination is rigidity of the abdominal muscles. It is true that the estimation of comparative regional rigidity is of the greatest value in differentiation, but frequently when an acute abdomen comes

under observation there is such marked wide-spread rigidity that no information can be obtained by palpation. Generally speaking, too, percussion is of little value in the differentiation of acute abdominal conditions unless there is a localised abscess, and inspection only shows that abdominal respiration is absent—a condition common to nearly all acute abdominal conditions. Inspection may indeed mislead unless one is alive to the variations in the appearance of acute abdomens. I have in my wards at present a boy, æt. 12 years, who was admitted with a gangrenous appendix in the 5 o'clock position, whose abdomen was flaccid and boat-shaped and gave no evidence of acute disorder. A rectal examination, however, made the diagnosis obvious enough. In my experience it is not unusual to find in children with a gangrenous 5 o'clock appendix a negative abdominal picture, and only the routine rectal examination will save us from overlooking what is perhaps the most dangerous form of acute appendicitis.

Another source of error is the quiescent period which occurs in the early stages of certain acute abdominal conditions. I have noticed it most frequently in cases of perforated gastric ulcer and perforated appendix. In such conditions it is the absence of signs and symptoms which makes differentiation impossible. I well remember a case of perforated gastric ulcer which I had the opportunity of seeing only two hours after the perforation had occurred. At that time the clinical picture of perforated gastric ulcer was so exact that no mistake was possible. Four hours elapsed before the preparation for the operation was completed, and by that time all the signs had disappeared except a loss of liver-dulness and some slight tenderness and rigidity in the right iliac fossa. The pulse-rate had fallen to 72 and the patient said he felt quite well again. I am sure that if he had been seen for the first time at that stage, it would have been impossible to have made a correct diagnosis, except from the history.

It is a commonplace for cholecystitis to simulate an acute appendicitis. Gall-stone patients are generally very fat, and this, of course, makes diagnosis more difficult, but in many cases a history of typical attacks of colic, and even of jaundice, may be obtained. Acute mucocele or empyema of the gall-bladder should generally be diagnosed, as the distended gall-bladder is palpable in most cases, but there is real difficulty in the differentiation between appendicitis and a cholecystitis that has resulted in perforation or gangrene with extensive local or general peritonitis.

Another common mistake is to confound a right-sided pyosalpinx and a 5 o'clock appendix, and if there is much matting in the pelvis differential diagnosis must be impossible. The presence of vaginal discharge or a history of long-continued pelvic pain or discomfort will of course be in favour of a salpingitis, and on the whole a pyosalpinx, even when ruptured, gives rise to less urgent symptoms than an acute appendix in the 5 o'clock position.

Other disorders of the female pelvic organs which may simulate appendicitis are ovarian cyst with a twisted pedicle and extra-uterine gestation. Both these conditions give rise to very urgent symptoms, and are associated with rigidity of the lower abdominal muscles and pain and tenderness below the umbilicus. But an ovarian cyst is generally big enough to be felt, and extra-uterine gestation is associated with such a train of symptoms, *e.g.* amenorrhœa, activity of the breasts, brown vaginal discharge and signs of acute internal hæmorrhage, that diagnosis is as a rule easy. I remember a case, however, which was a great puzzle. A girl, æt. 20 years, was seized with sickness followed by abdominal pain. The sickness soon ceased but the pain continued; thirty hours later the temperature rose to 101° F., and she complained of severe pain limited to the appendix region, and she passed several watery stools with blood in them. When I saw her two hours later the temperature was 101·5° F. and the pulse 80. She was flushed and the tongue was clean. Abdominal examination showed a rigid right rectus with a point of great tenderness at the level of the umbilicus, though she complained of severe pain in the right iliac fossa. *Per rectum* there was a large, soft, acutely tender swelling in the pouch of Douglas. A watery stool containing blood had just been passed. Soon after the pulse-rate rose to 96 and operation was decided upon. The appendix was normal, except for a small hæmorrhage, but a large right ovarian cyst was found, not twisted or inflamed in any way. After operation there was no more diarrhœa, but convalescence was retarded by an evening pyrexia, œdema of right leg and right side of vulva and purulent vaginal discharge.

I chanced on an unusual condition simulating

appendicitis last December, when a woman, æt. 49 years, was admitted with a history of acute abdominal pain occurring suddenly two days before. She had pelvic peritonitis, apparently of some duration, with a large mass above the pubes towards the right side and an indefinite inflammatory mass in the pelvis. Her temperature was 100° F., pulse-rate 96. At operation I found a sloughing fibroid of the uterus for which I performed hysterectomy.

It is not necessary to do more than remind you of the difficulty in differentiating between appendicitis and some cases of acute intestinal obstruction. So varied are the symptoms and signs in those conditions that it is impossible to lay down hard and fast rules for their differentiation. Experience, and experience only, will enable one to come to a correct diagnosis, though one may say in general terms that absence of abdominal respiration and the presence of muscular rigidity are in favour of an inflammatory lesion, while it is an almost invariable rule that *pain* is the first sign of acute appendicitis. This fact was of great assistance to me in the case of a school-boy, æt. 15½ years, whom I saw last March with acute abdominal symptoms. His illness commenced five days before with sore throat, and after a short period of malaise, pain in the abdomen came on without vomiting. When I saw him his temperature was 103° F., pulse-rate 124, and he looked very ill. He was complaining of general abdominal pain, but examination did not show any localised condition of abdominal inflammation. It was an instance of an acute abdominal condition, which, for want of a better idea of the pathology, one might call influenzal, and he soon recovered under medical treatment.

It is quite possible to mistake such a condition for appendicitis, as occurred in the case of a girl, æt. 10 years, whom I saw at the beginning of last year. There was a history that seven days before she had had a feverish attack with pain and stiffness of the neck. She was sick and was kept in bed for three days. On the day I saw her she was seized with sudden, severe generalised abdominal pain and vomited. Temperature reached 100° F., and pulse-rate 96. She looked extremely ill, but was not in pain. There was marked rigidity of the abdominal muscles below the level of the umbilicus, the epigastric angle being quite flaccid, and there was heat and bogginess in Douglas's pouch on rectal examination. Examination of the chest was negative. On opening the abdomen the appendix was found normal, but there were several ounces of clear fluid in the abdomen. The appendix was removed and the abdomen closed without drainage, and convalescence was rapid.

Very similar conditions are seen in cases of acute tuberculous peritonitis. A boy, æt. 7 years, came into hospital in September last with a typical acute abdomen and a history of a sudden attack of abdominal pain and vomiting. Rigidity and tenderness were so marked that a differential diagnosis was impossible, but as it was evidently a case for immediate laparotomy the abdomen was opened, a quantity of clear fluid was evacuated, and the peritoneum and omentum were found to be studded all over with minute tubercles. The ascitic form of tuberculous peritonitis gives, of course, the best results from the operative point of view, and, as one expected, this case did very well indeed.

A curious case illustrating the difficulty of differential diagnosis occurred in a boy, æt. 16 years, who, during the course of an acute pneumonia, was seized with sudden right-sided abdominal pain. In view of the concurrent pneumonia, his temperature and pulse-rate and general condition were of no assistance in diagnosing this new development, although the temperature, which had been falling, rose to 104° F. when the pain began, and we had to depend upon the local signs. These consisted of rigidity of right rectus with acute tenderness just below the umbilicus level, and a wide-spread resistance with fairly-well defined margins, resembling an intra-abdominal abscess. The condition proved on exploration to be a rupture of the sheath of the right rectus, as the result, I suppose, of straining to cough, with extravasation of blood. I closed the wound after suturing the sheath without opening the abdomen.

The mention of pneumonia reminds me of the necessity of differentiating in children between appendicitis and the abdominal pain which occurs sometimes in right-sided pneumonia or pleurisy. This matter was first brought prominently before the profession by the late Mr. Barnard, and many cases have since been recorded. It may not be possible in all cases to demonstrate the chest trouble at first, as the abdominal pain is an early

sign, but a diagnosis from acute appendicitis may be made from the facts that the temperature is too high (acute appendicitis rarely has a temperature over 102° F.), there is evidence of hurried, and perhaps of laboured respiration (*e. g.* movement of the alæ nasi), and the abdominal rigidity is less constant than occurs in an acute abdominal condition.

A condition that is exciting a considerable amount of interest just now is acute infections of the kidney, whether hæmatogenous so-called, or ascending. The pyelitis of pregnancy is generally an infection by the colon bacillus and nearly always affects the right kidney. Hæmatogenous infections affect either kidney, and are due to staphylococci, streptococci, or the colon bacillus. If the right kidney is affected the clinical picture is that of sudden, acute right-sided pain, not confined to the loin, but passing downwards into the right iliac fossa towards the pelvis. This is associated with a high temperature, rapid pulse-rate, rigidity of the muscles, and hyperæsthesia both in the loin and in the right side of the abdomen. Indeed, this hyperæsthesia is the best guide to the diagnosis, as even a retro-cæcal (11 o'clock) appendix will rarely give rise to so marked a tenderness in the costo-vertebral angle. Very often in the early stages there may be no abnormality in the urine, and no frequency of micturition, and this may lead one to overlook the kidney as a source of the symptoms. I have seen three or four of these cases in the last few weeks, and in one case particularly diagnosis presented many difficulties.

Before considering conditions simulating chronic appendicitis attention must be directed to two conditions which must be differentiated from appendicitis with localised abscess. Six years ago I had in my wards a middle-aged woman who had had pain in the right iliac fossa and signs of partial obstruction for an indefinite period, culminating in local pain and tenderness of some severity. On examination a fixed swelling was felt in the right iliac fossa, giving the impression of a mass of adhesions around an abscess in connection with the appendix. Laparotomy showed that the condition was one of hyperplastic tuberculosis of the cæcum, a disease which had formed the subject of a most instructive lecture by Professor Hartmann at the Medical Society of London a few weeks before.

The other condition is actinomycosis of the cæcum, and it happens that I have in my wards at the moment a woman, æt 47 years, upon whom I operated a fortnight ago for this disease. The symptoms differ, of course, from the ordinary appendix abscess in the relative slowness of development, and in the present case the abscess seemed to involve the skin much more than is usual in appendix abscess. The temperature and pulse-rate were normal, but in this connection it should be remembered that the temperature and pulse-rate are generally unaffected in appendix abscess if the abscess is of long standing and well shut off by adhesions.

Conditions Simulating Chronic Appendicitis.

When we consider the signs and symptoms upon which we form a diagnosis of chronic appendicitis, we shall be a little surprised to realise how much we depend upon pain. In many cases, indeed, of quiescent relapsing appendicitis, there are no signs whatever, and the diagnosis must perforce be made from the history of previous attacks, and the subjective symptoms of pain and perhaps of tenderness. But, of course, many disorders may give rise to pain in the right iliac fossa, and it is necessary to guard against the tendency to diagnose appendicitis in every case of right-sided abdominal pain.

Tuberculous glands in the mesentery, especially if they have reached the stage of caseation or calcareous degeneration, occur with some frequency in children and even occasionally in adults, and often lead to kinking of the intestine with, as a result, colicky pains of a severe and even agonising character.

In September, 1910, I was asked to see a girl, æt. 4 years, who had for the last three weeks had attacks of paroxysmal abdominal pain, severe enough to make her cry out. The pain was limited to the right iliac fossa, but there was no rigidity or tenderness anywhere. The bowels were regularly opened, the tongue clean, pulse-rate about 100. Laparotomy revealed a caseating gland in the mesentery of the ileum, causing a very definite kink of the bowel. The pain ceased at once after removal of the gland and has not recurred.

A boy, æt. 10 years, seen in November, 1910, had a similar condition, characterised in his case by pain in the right iliac fossa, with tenderness, but

without vomiting or constipation. Both these children had a strong family history of tubercle.

I operated upon a woman, æt. 29 years, three weeks ago, who was suffering from intestinal stasis. X-ray examination after a bismuth meal showed ptosis of the cæcum with high fixation of the lower end of the ileum with kinking, and the case was looked upon as an ordinary one of ileal kink. On opening the abdomen the faulty position of the ileum was found to be due to its fixation by three caseating glands in its mesentery.

My experience of these cases leads me to think that the typical symptom is constant pain in the abdomen with occasional exacerbation of paroxysmal colicky pain without vomiting or constipation, and one certainly does not expect to see stools characteristic of tuberculous enteritis, though doubtless the glands are in the majority of cases infected from the bowel.

Another condition that has to be distinguished from chronic appendicitis is colitis. Whether colitis is secondary to appendicitis or appendicitis to colitis is a matter for discussion, but it is quite certain that appendicitis can occur without colitis, and probably colitis can occur without the appendix being implicated. The cardinal symptoms are pain in the abdomen, not confined to the right iliac fossa, with tenderness over the course of the colon, constipation, and mucus in the stools. Many of these cases resemble a relapsing appendicitis very closely, and the greatest care is necessary to distinguish between them. Perhaps the most reliable method of diagnosis is the chemical and bacteriological examination of the fæces. Mucus is normally present in the stools, but only in minute traces, recognisable by chemical tests. If visible mucus is present it indicates catarrh in some part of the intestinal tract, *e.g.* (*a*) mucus without fæcal matter denotes inflammation of the colon, ulcerative colitis, or intestinal dyspepsia; (*b*) mucus intimately mixed with the fæces indicates catarrh of the small intestine or cæcum; and (*c*) mucus with much food residue denotes catarrh of the small intestine. The bacteriological examination generally shows that one or more of the normal bacterial groups are in excess and only occasionally are any new species found.

Of the difficulty in differentiating between appendicitis and such diseases as cholelithiasis and ureteric calculus I have no time to talk at length. Either condition may simulate appendicitis very closely. Of course an X-ray photograph and a cystoscopic examination should suffice to prove the existence of an ureteric stone, even if there are no marked urinary symptoms, and in cholelithiasis the attacks of pain are generally localised to the right hypochondrium, passing through to the scapular regions, with vomiting and flatulence, and as they quieten down a feeling of soreness is left which is almost chararacteristic. The examination of gall-stone cases should be undertaken with the patient in the sitting position. The surgeon stands behind the patient with his hands pressed firmly upon the patient's hypochondria. At each expiration the hands are pressed backwards and slightly supinated, so that after a few breaths the liver is brought down in inspiration directly upon the palmar surface of the examiner's right hand and fingers. If gall-stones are present the impact of the gall-bladder upon the resisting surface of the examiner's hand causes pain, which is evidenced by a characteristic catch in the breathing. This method will be found of great value in distinguishing not only between gall-stones and appendicitis, but also between gall-stones and affections of the kidney.

Duodenal ulcer has such a characteristic train of symptoms that it can hardly be said to mimic appendicitis, and with regard to gastric ulcer it would be more true to say that occasionally appendicitis simulates gastric ulcer rather than that gastric ulcer simulates appendicitis. There is a condition, however, of great importance which simulates chronic appendicitis very exactly. It is characterised by the formation of adhesions between the colon and the peritoneum lining the outer wall of the abdomen, which tend to cripple the peristaltic movements of the large intestine, and to give rise to pain and delayed passage of the intestinal contents. The condition is associated with enteroptosis of greater or less degree, and, in later stages, by the formation of an adhesion, binding the lower end of the ileum firmly to the right wall of the pelvis. It is probable that the first stage in this process is a tendency to prolapse of the cæcum into the pelvis as a result of the upright position in patients suffering from habitual constipation. Bands form between the lateral abdominal wall and the colon in Nature's effort to afford support, and these, while serving their purpose to

some extent, hamper the peristaltic movements of the colon and thus increase the stasis of its contents. This condition is naturally seen more on the ascending colon than on the descending, as the hepatic flexure is much less securely supported than the splenic flexure, and there is, owing to the force of gravity in the erect position, a greater tendency to stasis in the ascending than in the descending colon. The effect on the ileum is, I believe, a later development, and results from the fact that as the cæcum descends it drags upon the ileum, which has, at its termination, a very short mesentery. The effect of constant dragging on this mesentery causes a thickening, which eventually becomes a band kinking the ileum at its point of attachment. The effect on the patient's health is very insidious. Starting with the cardinal symptoms of pain in the abdomen and constipation, there are gradually added all the characteristic signs of toxæmia, *e. g.* foul tongue and breath, sallow skin, loss of weight, headache, lassitude, and so-called neurasthenic symptoms. The right kidney is often too mobile and cystic changes occur in the breasts. These patients soon find that the recumbent position eases their pain to a marked degree, and this fact, with their general loss of tone, induces a form of chronic invalidism. In the late stages the condition is readily recognised, but in the early stages it may be easily mistaken for appendicitis. I have seen many of these cases of late, and an account of a few may help to acquaint you with the ordinary signs of early cases.

In November, 1911, I saw a girl, æt. 22 years, who gave a history of irregular abdominal pain extending over a period of nine weeks. The pain was at first in the umbilical region, but lately it has always been in the right side of the abdomen and low down. There had not been any vomiting, but constipation was present habitually. She had a furred tongue, foul breath, and was losing weight. At the operation adhesions were found between the cæcum and ascending colon and the outer abdominal wall, but the appendix was free and healthy.

A woman, æt. 30 years, was seen by me in July, 1912, who gave a history of an attack of pain in the right iliac fossa in September, 1911, with vomiting and diarrhœa. In February, 1912, she had a similar attack, and since that time she had had nearly constant pain in the right iliac fossa and umbilical regions, relieved by lying down, but increased by taking food. She complained of wind and general lassitude. A condition of peri-colonic adhesions was found, affecting the cæcum and ascending colon, the appendix being unaffected.

A woman, æt. 35 years, who had had her appendix removed without relieving her symptoms, came to me in September, 1912, complaining of pain around the umbilicus and in the right side of the abdomen. Constipation was marked, and there was flatulence and nausea, but no vomiting. A well-marked ileal kink was found at operation.

A girl, æt. 19 years, was brought to hospital in September, 1912, with severe abdominal pain of twenty-four hours' duration with vomiting. When I saw her she had a temperature of 99°F. with a pulse-rate of 112. The tongue was dry and coated, but although she complained of great pain in the right iliac fossa, there was no rigidity or abdominal distension. The symptoms quieted down, and a fortnight later her abdomen was opened and a well-marked ileal kink was found, the appendix being healthy.

A woman, æt. 27 years, was admitted to hospital in April, 1912, with epigastric pain with flatulence after meals which had lasted "for years." For the last two years she had had repeated attacks of pain in the right iliac fossa with marked constipation. An extensive condition of adhesions affecting the cæcum and ascending colon was found at operation the appendix being healthy and free from adhesions.

A woman, æt. 27 years, has just left the hospital who gave a history of "indigestion" with nausea and "hunger pain" lasting for four years. She had an attack of pain localised to the right iliac fossa in June, 1912, which lasted fourteen days, and a similar attack in December, 1912. Her complaint on admission was of pain in the right iliac fossa, always present, except when she was lying down, with marked constipation, loss of weight and general malaise. An ileal kink resulting from a very firm band of adhesions was found at operation.

These cases will give you some idea of the difficulty of differentiating them from cases of a relapsing appendicitis. In nearly all the pain was of no great severity, and rarely was anything definite made out on examination. But you will notice that the pain was chiefly right-sided and that constipation was present. In a typical case of appendicitis the pain is frequently epigastric and constipation is

not a feature. Nor in appendicitis does one find the relief from pain on lying down; there is no loss of weight, and no evidence of toxæmia. But the diagnosis can only be made by watching the passage of a bismuth meal through the digestive tract by means of the X rays. Three or four ounces of bismuth oxychloride or carbonate are given in thin gruel and photographs are taken at intervals and investigations made through the screen. The photographs demonstrate very clearly the presence of ptosis of the colon, of stasis and of malposition or obstruction due to kinking by adhesions. As a rule at least two days and probably three are required for the investigation, especially if there is much delay in the large bowel. If one has a case of chronic abdominal pain and constipation associated with signs of intestinal toxæmia and evidence of intestinal stasis on X-ray examination, one is justified in advising an exploratory laparotomy, being sure that the stasis is due to organic obstruction, which no amount of medical treatment can cure.

May 12th, 1913.

Fœtal Membranes for Skin Grafting.—The amnion and umbilical cord, Sabella says, can be utilised to good advantage in skin grafting. Before cutting the grafts, he advises carefully looking over the membranes for any abnormality or diseased portion. Also question the woman from whom these organs are obtained as to her previous history. If such caution is observed there will be no chance of infecting the patient. After the organs have been found free from disease or abnormality they should be immersed in warm water to wash away any of the vaginal excretion which they have taken up. They then are placed in a bottle containing normal salt solution. By keeping them immersed in this and changing the solution a few times before the use of the organs they will remain alive for over seventy-two hours.

When the patient is ready for grafting the organs are dipped several times in a new solution of normal saline and then are cut up into pieces in any shape and size desired. The cord should be cut open and the blood-vessels scraped out. This is done so that the membrane which is to be used shall be free from any hindrance to early union. The surface of the cord and amnion which is to be placed on the ulcer should be the continuation of the inner surface of the cord. The jelly of Wharton in the cord is of an alkaline reaction, and therefore is easily absorbed, and ultimately the membrane covering the cord is left on the surface of the wound.

The surface of the parts to be grafted must next be inspected. If there are undermined edges the edges should be cut away until healthy skin is reached. Then the surface is carefully cleaned and disinfected. If there are any exuberant granulations they should be levelled off to the skin. Then the surface is thoroughly asepticised. All bleeding must have ceased, whether the wound is recent or granulating. If oozing of blood persists the wound surface must be covered by a protective layer of rubber tissue, of silver foil, guttapercha tissue, oiled silk, or such like material, over which sterile gauze is applied and the skin grafting put off till next day. When the bleeding has ceased the grafting should take place. The area to be grafted should be cleaned thoroughly first with peroxide of hydrogen which is washed off with a bichloride solution of 1 : 1000 or 1 : 10,000. The parts are next washed with decinormal salt solution. After thorough cleansing the grafts are placed over the wound carefully and smoothly. When the whole wound is covered with grafts, place over them oiled silk fenestrated so as to facilitate drainage. The next dressing should be ordinary sterile gauze and soaked in decinormal salt solution or Carrel's solution. Over this gauze soaked with decinormal salt solution a damper or rubber tissue should be placed, so that the moisture may not evaporate. Over the damper more sterile gauze is placed and ordinarily dressed. The first dressing of sterile gauze should be daily changed and soaked in normal salt solution. In order that the grafts be not moved during the daily changing of the dressings, Sabella places collodion around the fenestrated oiled silk. No antiseptic lotion should be used; only the saline solution. The same kind of dressings should be applied until healing is complete. Very large surfaces can be made to heal in a short time by this process. Sabella has used this method with great success.—*Journ. A.M.A.*, vol. lx, p. 1030.

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A CLINICAL LECTURE

ON

FRACTURED FEMUR.*

By C. GORDON WATSON, F.R.C.S.,

Assistant Surgeon, St. Bartholomew's Hospital; Surgeon to the Metropolitan Hospital and to St. Mark's Hospital for Fistula, etc.

GENTLEMEN,—I wish to bring to your notice to-day two cases of fracture of the femur. One case has been operated on to correct a vicious union, the other to secure union in an ununited fracture. In both cases I have secured good union and a correct alignment without shortening, by means of plates and screws. In the "Chronicles of Christopher"† you are advised if you would learn about fractures not to read books, but to visit the Law Courts, where you are sure to find a barrister brow-beating an unfortunate doctor on the subject, and further you are advised, when called to a case of fracture, to refuse to go until you have joined the Medical Defence Union and filed your bankruptcy petition. Underlying this humorous advice is a well of wisdom. Let me impress upon you as students never to lose a chance of examining a fracture, and *after* you have examined it, carefully to compare the physical signs and the radiographic appearances. Never lose a chance of inspecting fractures under treatment in the wards, and learn from them prognosis as well as treatment. In practice the patient demands prognosis; you must learn prognosis by constant experience in the wards while you are students and while it costs you nothing. Later on you have to buy experience. Let your prognosis be mental and based on cases you have seen, and not on books. Never volunteer a prognosis in a case of fracture, but have your answer ready, and let it be guarded.

* Delivered at St. Bartholomew's Hospital.

† 'St. Bartholomew's Hospital Journal,' 1911.

Finally make a note at the time of the prognosis you give, for memory sometimes sleeps.

I will now ask you to inspect this skiagram (Fig. 1). I hope it will never be your lot in practice to see another like it. You observe that the femur has been fractured in the middle third and that a vicious union has occurred. The fractured ends overlap and form an acute angle pointing outwards. This skiagram was taken when the patient was first admitted. Now look at this plate (Fig. 2). This was taken fifteen days after operation. You see that the fragments are now in line and kept in apposition by means of a metal plate and screws.

Now look at the patient, a boy, æt. 7 years, who was operated on eight weeks ago. You can see no deformity. There is no shortening of the limb.

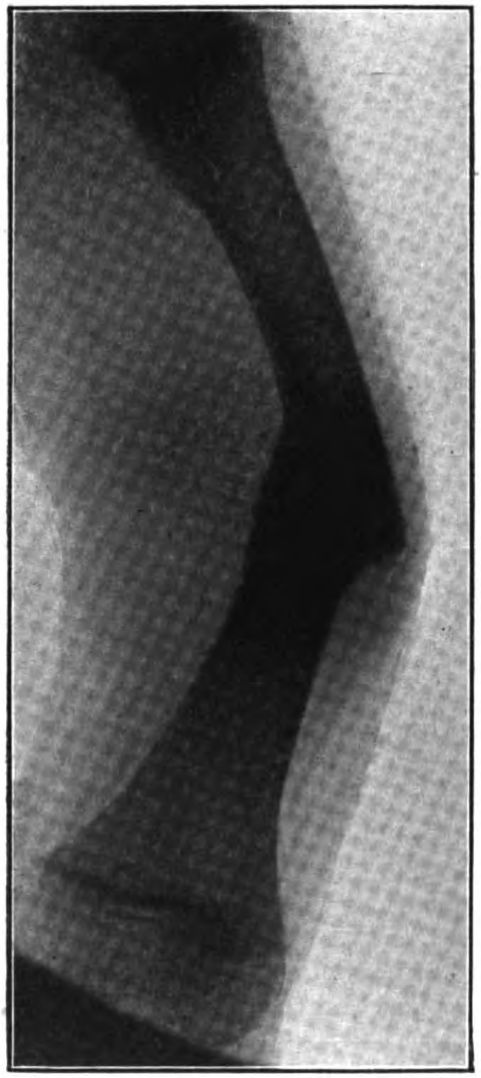

FIG. 1.—Vicious union with overlapping and considerable shortening.

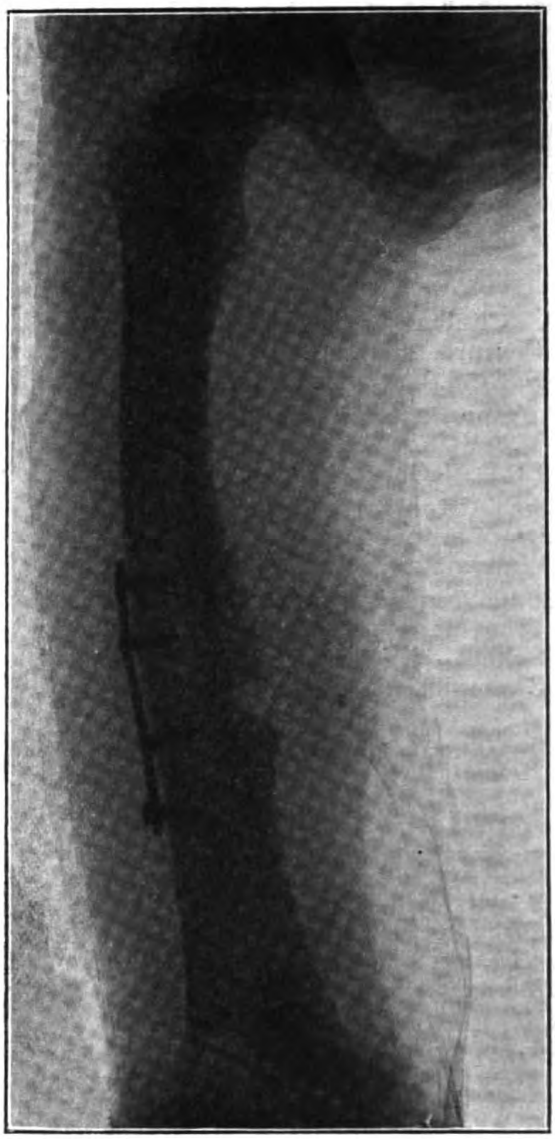

FIG. 2.—The femur in Fig. 1 fifteen days after operation.

He can walk well. On closer inspection you can see a scar four inches long. The muscles beneath the scar are not indurated.

Here is a skiagram (Fig. 3) taken eight weeks after the operation. It shows firm bony union. Be careful to note that this operation was performed, not for fracture, but for deformity following on a fracture of the femur, and due to incomplete fixation.

I believe that it is seldom necessary to apply internal splints such as plates or wire to a fracture of the femur in order to secure a good functional result. Occasionally, as in the boy before us, operation is necessary to correct a bad result. Had

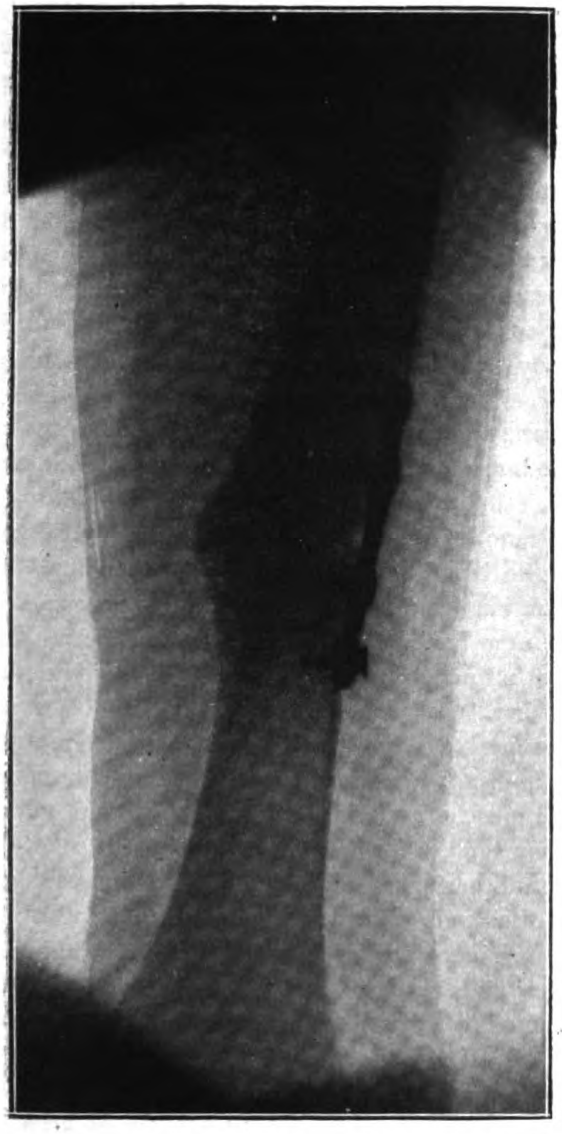

FIG. 3.—The femur in Fig. 1 eight weeks after operation.

this boy been treated in hospital from the first, probably no operation would have been necessary. In a few instances operation is necessary to secure union when, owing to the imposition of soft parts between the fragments, union has failed to occur.

The second case I am going to show you belongs to this class. This patient, æt. 48 years, fractured his femur in the middle third on August 18th of this year, and I show you the radiograph taken after the fracture (Fig. 4). Weight extension and a Liston's long splint were applied. At the end of five and a half weeks another radiogram was taken, which showed no union, a definite gap between the fragments and very little callus. I now show a radiograph taken two weeks after operation (Fig. 5). You observe the plate and the accurate apposition of the fractured ends. This

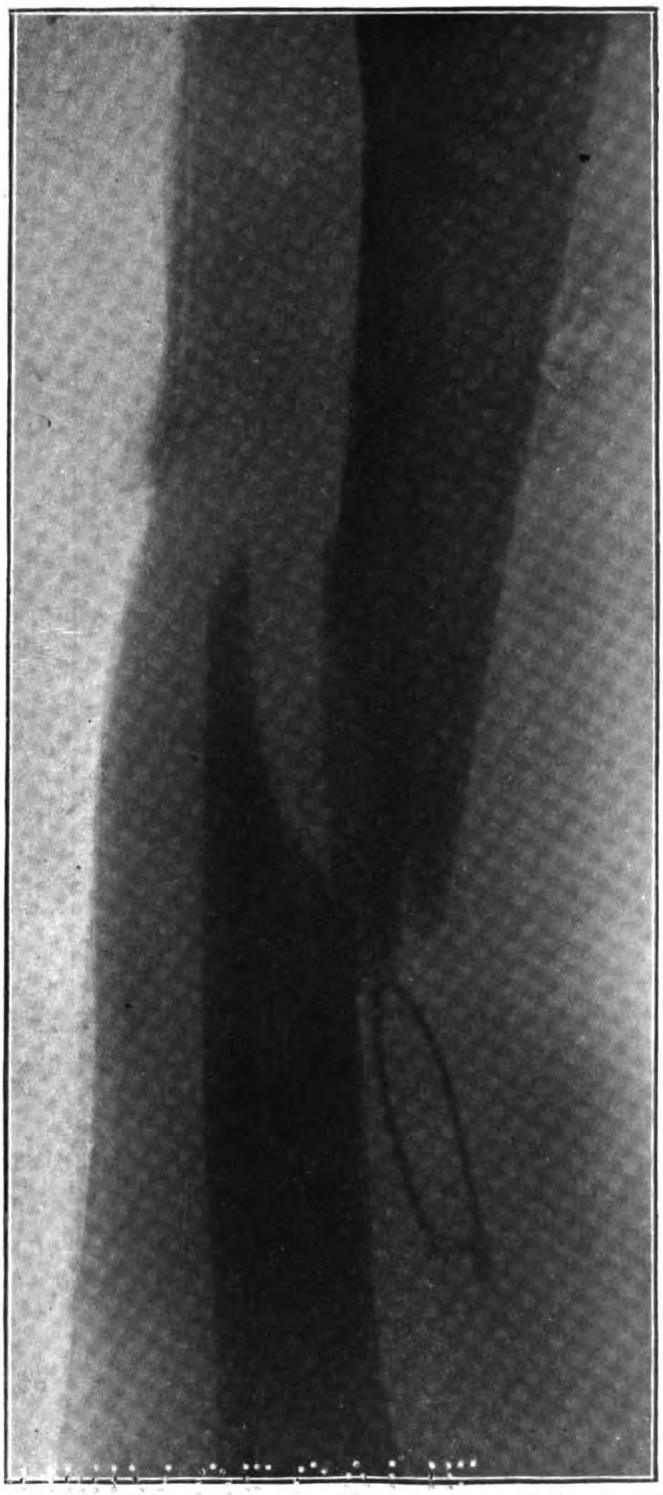

FIG. 4.—Non-union of splintered spiral fracture.

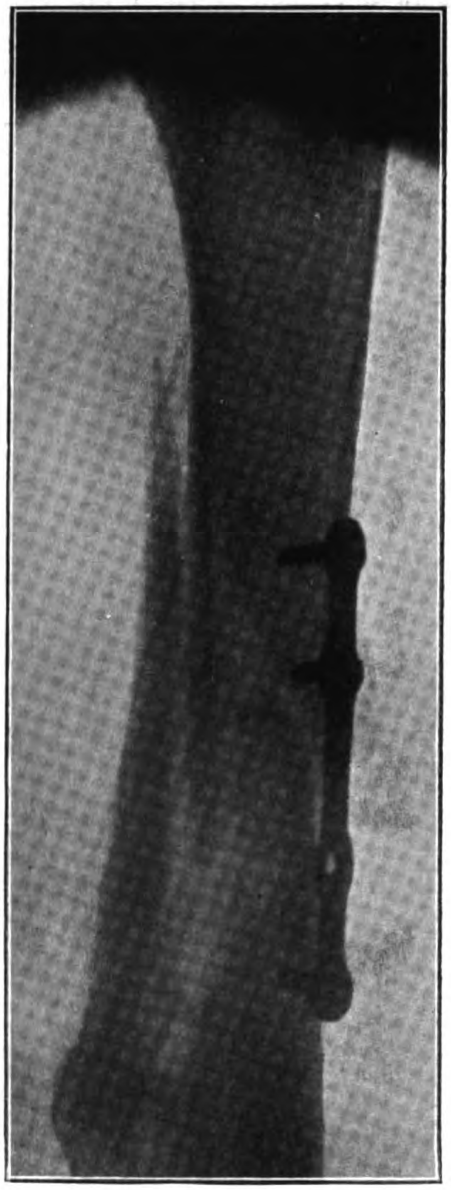

FIG. 5.—The femur in Fig. 4 fourteen days after operation.

patient has not yet commenced to walk, so that the case is not yet complete. The operation was performed seven weeks ago. He is having daily massage, and I expect he will be allowed to begin to use his leg in about a week's time. The boy commenced to walk with assistance at the end of six weeks.

I hope I have made it clear that these two cases were operated on, not as a routine, but for the special reasons of mal-union and non-union respectively.

There are those who argue that a perfect result after fracture of the shaft of the femur can be obtained only by operation and internal splinting. I do not deny that excellent results follow operation, but I know of, and have seen, bad results follow. Few operations, if any, require more detailed and careful technique to secure a good result. The most rigid asepsis is essential. It is my belief that the percentage of suppuration after operation on fractured bones is higher than in any other class of clean case. Remember that a foreign body is introduced into the midst of damaged tissues, and that a certain amount of oozing is bound to occur at the site of fracture, both from the bones and from the damaged muscles, and that the slightest flaw in technique may allow the entrance of organisms, which will inevitably flourish in the blood-clot round a broken bone. If suppuration occurs, not only is an unnecessary amount of new bone produced, but many muscle-fibres are ultimately displaced by fibrous tissue, and further, the plate or wire that has been used becomes a sequestrum and has to be removed. It is just as important to secure good function to the muscles as it is to provide firm and uniform bony support for them.

Another point I would like to make is that it is often very difficult and sometimes impossible to secure accurate apposition and good alignment even after cutting down and applying plates. Contracted soft parts have to be reckoned with as well as displaced bone fragments.

You must be very clean, very gentle and a good carpenter; you must check hæmorrhage very carefully indeed before you close the wound; you must close the wound with great accuracy, and you must ensure that the position you have secured is not altered by carelessness in moving the patient after the operation. You see there are many loopholes for failure, and on the other side we know that most fractures of the femur unite very well without operation, and that a good functional result can be secured with careful after-treatment.

Accurate alignment is not essential to a good functional result. Search out patients some years after fracture of the femur, X-ray them, and cross-examine them, and prove this for yourselves.

Although operation may be necessary to secure union in an ununited or mal-united fracture of the femur, it may not always be necessary to use plates or wire. If the line of fracture is oblique some

method of fixation is necessary to prevent subsequent displacement. If the line of fracture is transverse and non-union or mal-union has resulted with overlapping, then it may be possible to maintain end-to-end adaptation without plates, if the limb is carefully immobilised with external splints.

When the fragments overlap and the muscles have contracted, it is necessary to bring the broken ends out through the wound, and then with the ends impinging on one another to gradually replace them by pressure at the point of contact. This must be done without violence. Occasionally tenotomies are necessary before the bone can be replaced.

I now show you two radiographs of a transverse fracture of the femur. Looking at this one there appears to be perfect apposition and alignment. Now observe that in the other there is two inches of overlapping. The second view is from the side and shows that the lower fragment is displaced upwards behind the upper. The first plate was taken from the front, and owing to the fact that there was no lateral displacement the position of the fracture appears to be excellent. This point will impress on you the importance of examining fractures with the X rays from more than one direction.

I have said sufficient to indicate that although I show you to-day two cases of plating of the femur, I do not advocate this treatment as a routine, and it may be as well to refer to the lines of treatment which I do advocate. I do not propose to refer to fractures of the neck of the femur or to fractures in the immediate neighbourhood of the knee-joint, both of which require special consideration.

In putting up a fracture of the shaft of the femur an anæsthetic should always be given to secure complete relaxation of the muscles and freedom from pain during manipulation. If there is an open wound the following routine should be employed. Before any cleansing is commenced sterile gauze should be lightly packed into the wound, and when the wound is shut off the remainder of the limb should be cleaned, first with turpentine, ether or acetone, and then painted with 2 per cent. iodine over a wide area round the wound. When the skin round the wound has been cleaned the gauze plug should be removed, and the wound irrigated with saline solution. No antiseptics should be used. After thorough irrigation, the seat of the fracture should be examined, and any loose fragments removed. The wound is then temporarily covered up, and an extension apparatus applied to the leg up to the upper limit of the lower fragment. An assistant should then apply extension while the surgeon secures the best position possible for the fracture. The wound may then be closed, provided that the tissues have not been crushed by direct violence. In all cases of "run-over" fractures of the femur it is advisable not to close the wound. In the majority of motor-bus fractures dirt and micro-organisms are introduced, and *drainage is essential.* In these cases continuous irrigation should be employed from the first, and no sutures inserted. It is the usual practice in this hospital to apply a long Liston splint in combination with extension. If the fracture is compound the splint should be interrupted at the site of fracture to facilitate subsequent dressing. An X-ray photograph should be taken as soon as the limb has been put up.

In simple cases a splint is not essential for adults, provided that an efficient extension apparatus is used. Sand-bags may be used instead on either side of the limb to secure immobilisation. Sand-bags give facility for early massage, but a splint should always be used for children.

As I have already pointed out, the treatment of a fracture is not confined to the bones. It is most important to keep the muscles in good condition and to prevent adhesions. Massage not only serves this purpose, but is also most valuable for the relief of pain. Weight extension relieves the muscular spasm, and so is also an important factor in the relief of pain. Massage must be very gentle until union has occurred, and must not be allowed to produce pain. It should follow the course of the lymph flow. Some surgeons advise gentle mobilisation of the bones as well as massage to stimulate callus formation. It is certainly useful in cases of delayed union, and may possibly accelerate union in other cases. On the other hand, it may cause excessive callus formation, and may produce pain, more especially in children.

A few weeks ago a child was brought into the ward with a severe compound fracture of the leg, with extensive laceration of the soft parts. The child had been run over by a motor omnibus. Within twenty-four hours acute and spreading traumatic gangrene commenced, and spread from

the edges of the wound. The child was acutely ill, with a rapid pulse, high temperature, and frequent vomiting. I amputated through the middle of the thigh under spinal anæsthesia, but I was unable to save the child's life. I used spinal anæsthesia because I believe it produces less shock in these cases, especially in children. Subsequent cultures showed that there was infection with an anaërobe, the *Bacillus aërogenes capsulatus*, and that the organism was present in the blood. Amputation was too late. I have seen acute spreading traumatic gangrene after fracture of the femur in a patient run over by a motor 'bus come on within six hours of the accident, and a fatal result follow in spite of immediate amputation. Usually the gangrene commences from twenty-four to thirty-six hours after the injury. In cases of extreme laceration accompanied by contamination with road dirt, this is a not uncommon result.

Cleansing should be thorough, but I think antiseptics, at any rate in strong solutions, should not be used: they destroy what little vitality remains. The tendency should be to enlarge the wounds rather than to close them, and continuous irrigation should be employed. At the same time saline should be given liberally, either into the veins, subcutaneously, or *per rectum*, or by a combination of these methods. Cultures should of course be made, and I think horse-serum given subcutaneously may do good, and can do no harm.

No fixed rules can be laid down with regard to amputation. Both the general and local condition must be carefully considered. If gangrene commences at the margins of the wound, more especially if gas is formed in the tissues, I believe amputation is imperative, and even then it may not be possible to save the patient. The *Bacillus aërogenes capsulatus* is the organism most frequently met with in acute infective gangrene following injuries; in a few cases the bacillus of malignant œdema is found. In either case gas is usually formed in the tissues, and considerable swelling with surgical emphysema results. Not all cases of acute infective gangrene are due to anaërobes. The *Bacillus coli*, *Streptococcus pyogenes* and *Bacillus pyocyaneus* have also been guilty of producing the disease; the most fatal organism of all is the streptococcus pyogenes. Amputation must be performed clear of the infected tissues. You will think from what I have said that I am rather despondent about these cases. They are not all desperate, and I have seen amputation act like a miracle.

I want to impress upon you the extreme urgency of these cases. Unless you are familiar with them you will not realise that a limb, which, so far as the actual injuries concerned, is in no danger of requiring amputation, may very rapidly—almost in the twinkling of an eye—become a menace to the patient's life, and you may expect this if, after recovery from the initial shock of the injury, the rapidity of the pulse increases, the temperature remains high and perhaps rigors and vomiting occur, before any local signs of gangrene appear.

May 19th, 1913.

EYE-STRAIN IN EVERY-DAY PRACTICE. By SYDNEY STEPHENSON, Editor of the 'Ophthalmoscope. (London: The Ophthalmoscope Press, 24, Thayer Street.) Pp. 139. 3*s*. 6*d*. net.

It is a well-known maxim, and one which will bear some emphasis, that there are three common causes of headache in the following order of their importance: (*a*) disorders in the function of the digestive apparatus; (*b*) eye-strain in its various forms; (*c*) neurasthenia or nervous exhaustion, with or without associated anæmia. This books deals with the second of these causes. It is unfortunately too true that headache due to eye-strain is often diagnosed only after the whole gamut of coal-tar derivatives has been exhausted. It is the small things which often count; often worry, and certainly the ones which cause most trouble where the eye is concerned. The author draws attention to a ready and easily applied test, in order to decide whether a headache is due to neuralgia or eye-strain, viz. the instilling of a few drops of atropine solution for a few days. Dr. Weir Mitchell truly laid the profession under a great obligation by his strong insistence on a careful study of the eye in cases of chronic headache. It would be interesting to know what position headaches, acute and chronic, hold in the list of minor ailments which form the chief part of the average practitioner's work, and what proportion of such cases is due to eye-strain. A perusal of this book will amply repay the general practitioner.

ANGINA PECTORIS.

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ANGINA PECTORIS may be defined as pain at the præcordia brought on by exertion or emotion, but such a statement leaves much to be desired in precision. Is all pain at the heart induced in this way anginal, or conversely, is pain that comes on without either of these disturbing elements not anginal? In order to get over the great clinical differences that exist in the gravity of the results of such attacks of pain, it was at one time the custom to speak of those cases which were associated with obvious disease of the heart or great vessels as "true" and those not so associated as "false," but further experience showed that cases considered to be "false" sometimes proved fatal, and it had to be admitted that we possess no trustworthy means of recognising the most fatal of all forms of heart disease, that due to degeneration of the myocardium without enlargement of the heart or alteration of its sounds. It has also been suggested that in young persons—that, is under forty—the disease is false, but there are unhappily exceptions to what is doubtless generally true, nor can it be said that angina is always dependent upon recognised organic disease when it occurs in elderly people, although here again that is most often the case.

From a careful consideration of the facts it may be asserted that the pain is caused by a temporary want of proportion between the force of the heart and the resistance it has to overcome, but neither great weakness of the heart nor enormous increase in the resistance is necessarily accompanied by pain.

The best-known examples of abnormally high pressure are met with in contracting kidney, yet angina is only occasionally observed as a complication of this common disease, while on the other hand the blood-pressure may be low in quite well-marked cases of angina.

It is natural to assume that these should be the most serious, as where the resistance is not abnormally great the force of the heart must be greatly reduced, yet such cases do not always do badly. For example, E. D—, æt. 56 years, was seen first in 1903 complaining only of general weakness; he had had one attack of gout in the foot; no history of syphilis; he took no alcohol.* In May, 1910, he complained of tightness of the chest and pain radiating down both arms induced by exertion or excitement. Pulse-rate 60, blood pressure 125; physical signs negative. He was advised to give up tobacco, tea, coffee and cocoa, and given a mixture containing iodide of potassium 5 gr. and biniodide of mercury $\frac{1}{12}$gr. He improved, and two months later reported that he was keeping very well, but unable to exert himself. I have heard from him lately, and he tells me that he kept well until two months ago, when he had another attack which his doctor told him was *angina pectoris*, and he has been careful since to avoid exertion.

We should look for the causes of angina in two directions: first, those that weaken the heart by setting up disease of the aorta, the valves or the myocardium; second, those that induce wide-spread disease of the small vessels (arterio-sclerosis).

Disease of the aorta is chiefly the result of syphilis; it is possible that other infections occasionally determine it, and there may be a senile form of non-inflammatory origin. Syphilitic aortitis is proved by the clinical history, the early age of the patient, the presence of syphilitic miliary gummata in the walls and syphilitic lesions in other organs. Spirochætes have been found but rarely, as the disease has generally run its course by the time the organs come to be examined. A positive Wassermann reaction proves that the patient has had syphilis, but not that the aortic disease is syphilitic. Syphilitic aortitis is fairly common here, but not met with so frequently as, according to Stadler, it is in Leipzig, where he found it present in 211 out of 256 consecutive cases, of which in no fewer than 117 it was the cause of death!

Syphilitic disease of the aorta is a common cause of angina, even in comparatively young persons when it attacks the first portion, probably because it involves the coronary arteries or their orifices. According to Stadler, this affection may show itself in as short a time as five years after infection, though the average time at which the cases come under observation is fifteen to twenty years

* I have heard since that he was formerly intemperate.

after the initial attack; further, it may be due to congenital syphilis, so that the possibility of this condition as a cause of angina in young persons should not be overlooked. This is the more important, as if recognised in the early stages it is curable.

Of all infections acute rheumatism accounts for by far the largest number of cases of heart disease. A history of acute rheumatism is consequently met with in a large proportion of cases of angina, but, as is well known, there may be perfect compensation for the valve defect, and it is only when senile changes are superadded that anginal symptoms show themselves. J. H—, æt. 56 years, seen in 1911, had had rheumatic fever sixteen years before and two or three times since, but had felt no permanent ill-effects until the last year, when he had begun to suffer from attacks of pain and shortness of breath. The heart was not enlarged but there was a systolic murmur audible at the base, and I suspected that there was aortic regurgitation although I could hear no diastolic murmur. The pulse was 96, the systolic pressure in the right radial being 148, in the left 130. He had lost two stone in weight in the course of the last year. His urine contained a little albumen, with a few small granular casts but no sugar. He was rather a heavy smoker. A month later there was no albumen, and after two months he expressed himself as feeling "a lot better."

In this case the cause of the trouble was obviously the insufficiency of the heart, and abstinence from tobacco with heart tonics restored the balance.

My remark that there was probably aortic regurgitation in spite of the absence of a diastolic murmur is based on an experience of some years ago, when I observed the disappearance of the murmur and had my observation of its absence confirmed by many observers, while the subsequent *post-mortem* examination proved the existence of incompetent aortic valves. It has happened to many of us to see on the post-mortem table incompetent aortic valves in hearts where we had heard no murmur, and we may have blamed ourselves for overlooking it, but this case proved to me that there may be no audible murmur without any such special circumstance as that which happened in a case of the late Sir W. T. Gairdner's, where a polypoid vegetation had grown so as to form a ball-valve and restore the competence of the previously leaking cusps.

In another case of rheumatic angina the patient, a man, æt. 53 years, had had three attacks of acute rheumatism with no alteration of the heart-sounds. I had seen him three years before when his systolic pressure was 210 but his urine was free from albumen and the sp. gr. 1020. He was advised to give up whisky and tobacco and kept better until a month before his second visit, when his pulse was 60, pressure 169, heart-sounds normal, but the apex beat one inch outside the nipple line. Half a grain of erythrol caused headache, but 1 gr. of mannitol gave good results, and he reported that he was able to walk a mile twice daily without any pain. He gave up tea, coffee and cocoa by my advice and took Nativelle's digitalin ($\frac{1}{240}$ gr.) three times a day to strengthen his heart, keeping some doses of 2 ♏. of trinitrine for use if the pain recurred.

In this case the heart was evidently beginning to fail, and the angina was due to its relative weakness.

The heart may be weakened by over-strain, especially in the case of men no longer quite young, following sedentary occupations, badly fed and badly housed, but devoting their spare time to professional athletics, striving to break records in athletic competitions, bicycle racing, and the like.

Ambard believes that temporary hypertension is always accompanied by retention of chlorides, and that a chlorine-free dietary is indicated.

Dr. William Russell holds that there is a normal abdominal reflex which sets up vaso-motor spasm, and that this may be exaggerated by alcohol, tobacco, or excess of protein food; while there are individual variations, some persons being abnormally sensitive to these substances, and in them an attack of angina may be set up by exertion or emotion.

I am sceptical about this vaso-motor spasm being induced by alcohol, tobacco, or excess of protein food. I believe the first two weaken the heart without question, and the results of the endurance experiments of Irving Fisher and others afford some evidence that protein in excess, or perhaps purins, may have the same action, and I recommend abstinence from purins in the treatment of angina for this reason.

Rosenfeld, of Breslau, denies that alcohol stimulates the heart or raises the blood-pressure, but holds that it really weakens the heart, as indicated by the quickening of the pulse, while it probably

increases the viscosity of the blood, benumbs the vaso-motor centre, and favours calcification of the arteries. The evil effects of alcohol on the heart is very apparent in the weak, irregular heart which follows influenza, and my sphygmomanometer experiments confirm Rosenfeld's statement.

The causation of arterio-sclerosis is still a matter of controversy. As is well known, Huchard attributes it to a preliminary stage of persistent high tension, but this is disputed, and Stengel maintains that in the early stages the pressure is low; while even Josué and others, who attribute the arterio-sclerosis to hyperplasia of the adrenals, do not say that it results from hypertension, as in the experiments with adrenalin they found that the simultaneous administration of cholin, which prevents the hypertensive effect, does not prevent the production of arterio-sclerosis, so that they regard the changes as due to a specific action on the walls of the blood-vessels.

Aufrecht agrees with Stengel in believing that in the early stages the pressure is low, and E. von Neusser and Joseph Wiesel do not accept Josué's view that arterio-sclerosis results from hyperplasia of the adrenals, as Landau and Wiesel were unable to confirm the association, or to find that these organs are hypertrophied in chronic nephritis or in any renal disease.

Aufrecht believes that arterio-sclerosis begins as an inflammation starting from the vaso-vasorum, but the cause is obscure, as out of forty-five cases only seven gave a positive Wassermann reaction, and yet syphilis is the only certain ætiological factor.

Others believe that it is the consequence of alimentary toxins which find their way into the circulation in consequence of the failure of the protective agencies, especially in the liver, and alcohol is blamed for this. This is possible and even plausible, but, like all the allied theories of alimentary toxæmia, rests on no certain evidence.

Nearly all modern writers on angina recognise the influence of tobacco as a cause of attacks, although there is some difference of opinion as to whether it is capable of inducing an attack in a sound heart, or whether such attacks should always be taken to indicate some degree of organic weakness. I incline to the latter opinion, as experiments with tobacco do not show that it has any very marked effect upon the circulation when the heart is normal. Hesse made a number of observations upon medical students and hospital patients, taking the pulse-rate and pressure after one, two and three cigars smoked in succession.

In seventeen out of twenty-five cases there was a slight rise of frequency and pressure, in five no increased frequency, in three a slight fall of pressure with increased frequency. These changes were less marked in the young, but no difference was observed between those used to tobacco and those who were not.

My own observations gave very similar results. If tobacco accelerates the pulse-rate it would obviously be an efficient cause of angina where the aorta is weak, and this is much more likely to be the case in elderly than in young persons. I have repeatedly observed the greater susceptibility to cardiac pain after excessive smoking in persons over fifty, and without exaggerating the meaning of this as implying any very serious organic disease, it is in all probability dependent upon senile changes, which are common enough after fifty. A man who is a heavy smoker will find as he is approaching sixty that fast walking after smoking three or four pipes of tobacco will bring on sharp pain at the heart, and that by moderating the use of tobacco he keeps well.

Anything that weakens his heart, such as an attack of influenza, will increase his susceptibility, and may compel him to give up tobacco altogether until his heart has recovered. If this is often true of those whose hearts are fairly sound, it is much more likely to be the case in persons whose hearts have been weakened by acute rheumatism or syphilis or other infective processes.

Angina is often met with in elderly men who have albumen and sugar in their urine, slightly dilated hearts, enlarged livers, and whose habits in regard to alcohol have been more or less free. Abstinence from alcohol is followed by marked improvement, or in younger men by complete recovery. For example, F. B—, æt. 38 years, consulted me in December, 1899, for pain in the heart; he had had acute rheumatism five years previously; his heart-sounds and area were normal; urine showed a dense cloud of albumen and a little sugar; liver slightly enlarged. At that time he drank freely, but he became an abstainer, and is now (1913) perfectly well. His heart may have been weakened by the acute rheumatism but

there is no evidence of it, and I believe that its weakness was due to the effect of alcohol. I agree with Rosenfeld that alcohol does not strengthen the heart, but weakens it. This is very noticeable in the heart weakness that follows influenza, where the pulse is often irregular and intermittent and exertion is followed by feelings of faintness or exhaustion. In this condition it is natural to try alcohol as a remedy, and I have used it myself and seen many cases in which it has been ordered, but the effect is distinctly bad. I recognise the good effects of alcohol in the earlier stages of influenza and in other infections, typhoid fever and pneumonia for example, but I have never allowed it to be used as a routine remedy, and, therefore, my testimony to its utility should have greater weight; I do not believe the benefit is due to its action as a heart stimulant, but to the specific antitoxic effect which it undoubtedly possesses, as we see so very plainly in the treatment of snake-bite.

The influence of strain in developing angina in a weak heart was shown in the case of a colleague, æt. 59 years, who had for five years or more known that he was suffering from chronic Bright's disease, but had not exhibited any signs of high tension—in fact when the systolic pressure was measured in 1911 it was only 138. About two months before his death he very unwisely lifted a patient from the operating table, and from the date of this over exertion began to suffer from attacks of angina. In spite of warnings he continued to work, and in the morning of the day of his death did five operations, ending by performing artificial respiration for a case that had stopped breathing. This was the final effort. What actually occurs we do not know. Is the pain caused by stretching of the myocardium or of the wall of the first part of the aorta (Clifford Allbutt)? One would suppose that effort would stretch first the right side of the heart, and it is difficult to see that the "first part of the aorta" can be implicated at all during this stage, but after the respiratory muscles relax and the heart begins to beat violently and quickly the aortic pressure would rise, and it may be at this time that the pain starts. The fact that emotion which quickens the action of the heart without altering the respiratory movements is a potent cause of angina undoubtedly gives weight to this view. External cold is often capable of inducing an attack, in all probability in consequence of the contraction of the cutaneous vessels. In the case just mentioned there was a cold north-easterly wind blowing on the morning of his death, and when he arrived at the hospital he was "doubled up" with pain which he attributed to the cold, and it was not until he had given himself a hypodermic injection of morphia that he was able to begin his operations.

Comparatively slight importance should be attached to high blood-pressure in the production of angina. In fact so long as the pressure is high there will probably be no angina because the heart is strong, but as it fails then the increased peripheral resistance, whether temporary, as the consequence of vaso-motor spasm from external cold or permanent from arterio-sclerosis, becomes a factor to reckon with although their action is not identical. Vaso-motor spasm precipitates an attack, but arterio-sclerosis tires out the weakened heart, and renders it more liable to an attack from exertion or excitement.

The preventive treatment of angina consists in the avoidance of all those things that weaken the heart, and when it is weak, in the use of cardiac tonics, good food and a hygienic mode of life.

The prevention of attacks when the liability to them exists lies in avoidance of sudden exertion, excitement, overloading the stomach, alcohol, tobacco, tea, coffee and cocoa, animal purins and chilling of the surface of the body, especially by exposure to cold winds.

The drugs that are of most service are digitalis or Nativelle's digitalin, or where these disagree, strophanthus or Merck's strophanthin, taken regularly for some weeks. The tincture of digitalis or strophanthus in ten-minim doses or gr. $\frac{1}{10}$ of Nativelle's digitalin or gr. $\frac{1}{500}$ of Merck's strophanthin three times a day.

During the attacks trinitrin in 2-minim doses dissolved in water is better than chocolate tablets or than nitrite of amyl. Two or three doses of trinitrin can be carried in the waistcoat pocket in separate small bottles. Its action is more prompt in solution and its effect is more lasting than amyl nitrite. Erythrol gr. $\frac{1}{4}$ and mannitol gr. 1 have proved useful in certain cases, but I give the preference to those first-named.

Where the patient is obese the diet should not only exclude purins, but be chlorine-free, no common salt being used in cooking or at table and salt-free bread should be prescribed.

For syphilitic aortitis, biniodide of mercury gr. $\frac{1}{12}$ and iodide of potassium 5 to 10 gr. are still the best treatment. Stadler says that salvarsan has been often followed by fatal results, but he thinks that these cases should be treated in hospital or in a nursing home.

Inhalations of oxygen have been sometimes found to give relief, but the gas should be warmed by passing it through hot water. For this purpose Messrs. Burroughs Wellcome & Co. supply a convenient apparatus.

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THE AUTOPLASTIC OVARIAN GRAFT AND ITS CLINICAL VALUE.

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A YEAR ago I showed before the Birmingham Branch of the British Medical Association a patient upon whom I had performed heteroplastic ovarian grafting, *i. e.* the transplantation of the ovary from one patient to another. Shortly after reading this paper an opportunity occurred of trying the effect of an autoplastic graft; in other words, the transference of ovarian tissue from its normal position in the pelvis to a site elsewhere in the same subject. The result of this experiment has so far been entirely successful.

The details of the case* are as follows:

R. G—, æt. 29 years, a patient of Dr. Branday, of Handsworth, was sent into the General Hospital, Birmingham, on April 3rd, 1912, with a history of repeated attacks of pelvic inflammation. The woman had been confined to bed for one or two months with severe pain during each year for the three years previous to admission. Dysmenorrhœa had been severe for the last two years, following the birth of her last child. During this time the catamenia had been regular.

On admission the patient looked anæmic and ill and was considerably wasted. Bimanual examination showed that the uterus was fixed by adhesions, and that a very tender rounded swelling was present in the region of each uterine appendage, occupying the corresponding posterior quadrant of the pelvis.

On April 4th, 1912, I performed laparotomy through a vertical four-inch incision over the right rectus muscle, and found both uterine appendages in a condition of severe chronic inflammation, in fact, far too advanced for any conservative measures to be employed. The right ovary was the seat of a suppurating lutein cyst, of the size of a small orange, and was firmly adherent to the corresponding Fallopian tube. The left ovary was strongly

* Shown before the Birmingham Branch of the British Medical Association, February 13th, 1913.

bound down to the left tube and had to be removed with the latter, which was also chronically inflamed. The uterus, although presenting numerous adhesions on the peritoneal aspect, appeared normal, and was left *in situ*. Both uterine appendages were removed, but before the abdominal wall was closed a portion of the left ovary was grafted into the latter. The healthy half of the organ was cut into minute fragments and these were scattered into the subperitoneal connective tissue and within the rectus muscle and rectus sheath. Care was taken never to remove the grafted material from the body fluids. Until it was required it was allowed to remain in Douglas's pouch. The abdominal incision was closed in layers and no antiseptics were applied to the wound.

The patient made an uninterrupted recovery, and the incision healed by first intention. No undue tenderness has been noticed by the patient in the line of the incision either during convalescence or since.

The after-history of the patient is quite satisfactory, and it appears certain that some of the ovarian grafts have established themselves and are functionating. I heard no more of her until the end of January, 1913, when I communicated with Dr. Branday, and he very kindly made arrangements for me to examine her. The patient told me that she had not suffered from any "flushing," palpitation, or nervous phenomena, that are usually associated with the artificial menopause. The most important evidence, however, consists in the menstrual history since operation. The first period occurred from April 6th to 16th, 1912, commencing two days after the operation, when it was normally due. The next period began two months after leaving the hospital, and lasted ten days. The following period occurred three months later, lasting six days. On January 12th, 1913, a normal period occurred, and on February 13th uterine hæmorrhage again commenced. The last three periods have occurred at monthly intervals, and it appears therefore that regularity is established. During the catamenia rather severe headache has been present, but this has not been noted at other times. No dysmenorrhœa has occurred, and there has been no tenderness in the abdominal scar during the periods.

Inquiries as to the sexual life since operation showed that no dyspareunia is present, and that the sexual functions are exercised in a perfectly normal manner.

Remarks.—The case has not been recorded until almost a year after the operation, in order that a fair estimate might be made of the result. So far, this is as satisfactory as could be desired. Various criticisms may, of course, be urged against the acceptance of the graft as the cause of success. It is well known, for example, that even after double oöphorectomy, menstruation may continue for a few months. It rarely continues, however, as long as ten months after the operation, and, furthermore, the periods progressively decrease in duration. In the present case there is certainly no diminution, and, if anything, the flow is excessive. Again, it may be asked whether the uterine hæmorrhage is really of the nature of menstruation. The periodicity and the association of molimina in the form of headaches certainly strongly favour this view. The point may be settled by an examination of the menstrual fluid, and the demonstration in the same of an excess of calcium salts and absence of fibrin. This investigation will shortly be undertaken. Another criticism may be offered in the suggestion that all ovarian tissue was not removed from the pelvis, and that it is particularly easy to leave a portion or an accessory ovary when numerous adhesions are present. In the case here recorded I am inclined to think that this fact need not be entertained. The uterine appendages were removed without difficulty, the right ovary was replaced entirely by an abscess-cavity, and the left organ was adherent to the corresponding Fallopian tube only and not to the neighbouring viscera. At the time a careful search was made for any displaced ovarian tissue or accessory ovary, and none was seen as far as macroscopical appearances were concerned.

Literature.—The subject of ovarian grafting is a fascinating one, and I am convinced that it can take a very definite and important place in gynæcological surgery. The literature upon the subject is scattered, but is yearly increasing in amount, and sufficient authentic cases are now recorded to prove that autoplastic grafts can be successfully made, and that for a time at least they will functionate. The work of Scott Carmichael of Edinburgh (1), Higuchi of Tokyo (2), Nattrass of Melbourne (3), and others has proved that in animals, especially in the dog and rabbit, it is possible to transplant

ovarian tissue, and that the grafts are usually successful in producing the specific internal secretion. Nattrass's (3) experiments are extremely important in this respect. He was able in the case of the rabbit to transplant ovarian tissue into the subcutaneous tissues, and on to the peritoneum, kidney, spleen and periosteum. At first fatty degeneration of the stroma cells takes place, and it appears that this occurs in inverse ratio to the vascularity of the tissues into which the graft is made. When the tissue is examined at a later period, absorption and repair of the pathological products has occurred and the production and maturation of Graafian follicles takes place. That the transplanted organ exerts its influence over the sexual function has been proved by the same author in the case of a fox terrier bitch, in which œstrum was observed several times, and the ovary was found to contain large Graafian follicles 370 days after the grafting had been performed.

Evidence is not lacking, moreover, that grafting may be successfully accomplished in the human subject. Franklin H. Martin (4) has published the histories of six patients who continued to menstruate after transplantation of the ovaries, and Davidson (5) has recently recorded before the Edinburgh Obstetrical Society the case of a patient who began to menstruate three months after the operation and has continued to do so since, without either local or pelvic pain. To those interested in the subject the valuable monograph of Sauvé (6), published in 1909, may be recommended as containing a full account of the history and early experiments in this field of investigation.

"Seedling" grafts.—In almost all cases recorded in the literature, with the exception of those of Scott Carmichael, the graft has consisted of the whole or a very large portion of the ovary. I am inclined to think that much greater success will attend the use of small portions of tissue, as is done in the case of the thyroid and other glandular tissues. Because the ovary is a small gland, it does not necessarily follow that it can be lifted bodily and placed in a new position. The vitality of the tissue is much more likely to be maintained if "seedling" grafts are made. Furthermore, the chances of success are proportionately increased by the number of grafts made.

Site of implantation.—As regards the site of implantation, my own preference is for the rectus muscle and subperitoneal tissue. The reasons for this are as follows: In the first place, a good vascular supply is assured; secondly, the grafting may be most easily accomplished in this position, and involves very little increase in the time of the operation; lastly, should the graft require removal at any time, either from late suppuration or the development of a neoplasm, it may be reached easily and without the risk of opening the peritoneum. Further observation, of course, must show in what proportion of cases it is necessary to interfere at a later period for morbid changes occurring in the graft. So far as published records point, this eventuality is extremely slight.

Indications.—The indications for ovarian grafting will naturally be limited. One of the most important uses for the operation is indicated in the case recorded earlier in this paper, viz. as a conservative measure when it is necessary in a young woman to remove both uterine appendages for old-standing inflammatory disease without having to sacrifice the uterus.

Opinions will undoubtedly differ as to the proportion of cases in which this is possible. Undoubtedly a large number of cases occur where the uterus has to be removed with the appendages. Nevertheless, it is remarkable sometimes how rapidly and completely adhesions disappear when the inflammatory foci in the tubes and ovaries are removed. Furthermore, observations that I have recently made upon the bacteriology of the uterus in cases of chronic pyosalpinx have led me to believe that the endometrium is as a rule sterile, and therefore I have no compunction in leaving the uterus in such cases, especially when it may be possible to graft ovarian tissue into the abdominal wall. Another indication which I put forward as a suggestion is the possibility of transplanting fragments of ovarian tissue in cases of severe dysmenorrhœa. Instances of most intense menstrual pain undoubtedly occur where the source of discomfort lies, not in the uterus, but in the ovaries. Curetting and drugs rarely cure such patients, and if an examination is conducted during the height of the attack, the ovaries can frequently be palpated as extremely tender bodies occupying the posterior quadrants of the pelvis. In more than one case, oöphorectomy has been, and is still, practised for this condition, and I know of instances where pan-hysterectomy has been performed. A cure is undoubtedly effected thus,

but may not it be possible to bring about the same result without unsexing the patient, by substituting ovarian grafting? Many of these patients are young women, and it appears more rational to attempt a cure by conservative measures rather than acknowledge the failure of our art by subjecting the patient to a radical operation in which she loses those very organs which produce the hormones characteristic of her sex.

In conclusion may be summarised those points which I regard as essential to the success of a grafting operation.

(1) Absolute asepticity and the avoidance of strong antiseptics which would destroy the vitality of the tissues. The apparent contradiction to this maxim in the cases of chronic pyosalpinx and salpingo-oöphoritis is met by the fact that the pus in such cases is usually sterile and the tissues may be treated as surgically "clean."

(2) The employment of minute or "seedling" grafts.

(3) The presence of a good vascular supply in the tissue used as the bed for the graft. Muscle is entirely satisfactory for the purpose.

(4) The ovarian tissue should be left in contact with the body fluids within the peritoneal cavity until it is required for the purpose of the grafts. In the case recorded in this paper, the ovary was placed in Douglas's pouch until the time arrived for closure of the abdominal wound.

The object of this communication will have been attained if it serves to draw attention to the investigations of the various workers in this field throughout the world, and to the possibilities that underlie the wider adoption of the principle. Laboratory reseaches have already demonstrated the success that may be obtained, and it now remains for the gynæcological surgeon to apply these results, to glean fresh details from the after-results of his operations, and to formulate indications for the future employment of this branch of our art.

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May 19th, 1913.

SOME REMARKS

ON THE

DIAGNOSIS AND TREATMENT OF SKIN DISEASES IN SCHOOL-CHILDREN.

By FREDERICK GARDINER, M.D., B.Sc., F.R.C.S.E.,

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In the study of skin eruptions in school-children I have for some years noted that errors could have been avoided if the observer had kept in his mind certain elementary facts. They may be roughly grouped under history, localisation, appearance of eruption, complications and treatment. Only the more common skin diseases are considered here, although it is to be remembered that school-children may suffer from rare conditions from time to time.

Last century all skin manifestations were regarded as the result of humours in the blood; after that the pendulum swung round, and the teaching was that they were all caused by some local irritation. Now the saner view is held that although many eruptions may have a local cause, still their permanence and severity may be determined by the general health of the individual. A well-trained observer will invariably take a wide view of each case, study the physique of the child, the dirtiness or signs of neglect and the state of the clothing. Sometimes children suffer from too much scrubbing, and I have recently pointed out that the modern soap may be useful for clothes, but in many cases is harmful to the skin. Carbolic soaps and lotions often prolong or aggravate an eruption. I well remember a child of about six years of age coming to the out-patient department with a well-marked dermatitis of the hands, which was defined exactly by the circle round the wrist where the sleeve came down to, and in which the history was that she had been playing with the soap when the mother was at the wash-tub.

It is of interest to have some idea of the comparative frequency of the various diseases. A rapid survey of the statistics of the Skin Department of Edinburgh Royal Infirmary shows that impetigo contagiosa takes precedence, closely followed by

ringworm. Pediculosis, scabies, dermatitis, seborrhœa and psoriasis are a little more than half as frequent; tuberculosis and alopecia are represented by a still smaller number.

Impetigo contagiosa.—A very recent origin, a common history of others in the family being affected and an essentially superficial type of eruption, these facts should be imprinted in red letters on the memory, and then the frequent mistakes in diagnosis would not occur. No one should be misled by the parental remark that "it comes out every spring," and that the child's "blood is out of order." Pediculosis capitis is present in a large percentage of cases. and this may have something "to do with the spring." The classical stuck-on crusts and absence of depth in the lesions, with the localisation to the scalp, face and hands are, with the above history, quite sufficient for diagnosis.

As regards treatment, the rule is, to use a weak antiseptic ointment and apply very often and in large quantities. One finds many instances of persistence even with this treatment, and then the fault generally lies in not attending to the pediculosis. This is readily overcome by treating the head with paraffin oil. Still another cause of persistence is the presence of seborrhœa; this is diagnosed by the scaliness of the scalp and the redness and exudation behind the ears, and a variation in treatment by the addition of ten grains of sulphur to the ounce of ointment is of value.

Ecthyma.—Here we have to deal with the underfed or at least the debilitated child, and to ensure a rapid cure attention must be paid to this. The history may be of longer duration and very often the lower limbs are more markedly affected. The lesions are deeper, and generally there are unhealthy ulcers. In considering the lines of treatment one must remember that the lesions are due to the action of a weak organism on an unhealthy skin.

Pediculosis.—When thoroughly treated this affection is readily removed, but occurring as it does in a patient who generally has an unsatisfactory home, it is sometimes rather difficult to eradicate unless proper disinfection of the clothing and bedding can simultaneously be carried out, so that many cases persist for long periods. It is well to be charitable and not always look contemptuously at the afflicted; I am quite certain that there are types of hair which are better breeding-grounds for the pediculi, and as even in adults one regards the anæmia, sometimes intense, to be often *ante hoc*, so the same may be the case in children. On the scalp the best places to look are at the occiput and the region of the ears. It requires careful observation to identify their presence in slight cases, and it is these that give most trouble. It is a good rule to examine the scalp in all facial skin conditions; even if we only find seborrhœic scales, still, the application of remedies for this may lead to success where otherwise failure would have ensued. On the other hand one regularly sees students and practitioners mistake the ova for scales. No matter what the social status of the patient, if the scalp is very itchy, look for the ova, and one or two may only be found in the sites mentioned.

Pediculosis pubis affects the eyebrows and eyelashes in young children.

In searching for pediculosis corporis look at the clothing for the insect itself and examine the lanugo hairs with a lens for the ova.

Soaking the scalp with paraffin oil for thirty-six hours is the best treatment for pediculosis capitis. For pediculosis corporis and pubis the inunction of mercurial ointment and frequent washing is to be recommended unless in places like the eyelashes, where one can cut off the hairs and ova together.

Scabies.—This condition may go on for months, but the important point in the history is, that on careful examination some other members of the family will almost invariably be found to be affected.

In this connection it is not enough to take their word for it, but each individual must be examined. What often suggests the diagnosis is the appearance of scratched lesions on the arms and legs often combined with pustulation. The burrows on the fronts of the wrists, between the fingers, etc., should then be looked for although they may not always be clear. Urticaria is at times a complication of this disease, and when severe it sometimes dwarfs the scabies and leads to a mistaken diagnosis. Treatment should be thorough but of brief duration. If the sulphur ointment is used for more than three days at a time the resulting dermatitis is often more severe than the original disease; rather then give a few days' rest before repeating the inunction if the first trials have not been curative. Relapses are generally caused by want of attention to the effective disinfection of the clothing and bedding, or by overlooking one

individual in the family who may have only a mild attack.

Ringworm generally can be traced to some infective source. On the scalp the duration is long, but tinea corporis may also persist for months and may be very wide-spread. The ultimate diagnosis rests often with the microscope. On the scalp one should choose for this purpose the short broken or bent hairs, while on the body the scales at the edge of the suspected patches should be taken. There are occasions when not many broken hairs are seen, and yet when the scales are removed by forceps they show adherent hairs underneath. Again the hairs may be broken so close to the mouth of the follicle that a comedo extractor is necessary to remove them.

When soaked in liquor potassæ and examined under the microscope the scales show mostly mycelium, and in the hairs the spores mostly predominate.

Favus is a still more chronic affection, and the mousy odour, the dry hay-like hairs and the baldness and scarring are the chief features. The hairs most likely to show the mycelium under the microscope are those that come out with a swollen root when epilated. Scutula are not always present, but are diagnostic when seen. They, conversely to tinea scales, show mainly spores.

Alopecia areata.—In children this must be practically regarded as an infective condition. The "exclamation" hairs and the microscope distinguish it in most cases readily from ringworm, but there are quite a number which require most careful differentiation. A late diagnostic sign which is of great value is that the fresh hairs come in white and atrophied. The thinning of the scalp as a whole over the patches is also noteworthy. A variety is seen every now and then where the hair is broken off close to the mouth of the follicle and so simulates black-dot ringworm, but when attempts are made to epilate, these come out readily and reveal a long hair, not a stump. Pediculosis often aggravates and spreads this disease, so that when present it should be promptly treated. In early stages, where the areas are comparatively small, painting once a week with pure liquid carbolic acid gives very good results.

Seborrhœa and seborrhœic dermatitis date in many instances from infancy. The localisation is of the greatest importance. A scaly condition of the scalp may or may not be present. The flexures behind the auricles, and the auricles themselves, the neck, the sternal and interscapular regions and the flexures of all the joints, but especially the elbows and groins, are the common sites. The name of "intertrigo" is still regularly used by some writers, but if one notes this distribution accompanying a scaly eruption elsewhere the lines of most successful treatment will then be found to be that of seborrhœa. As illustration one may mention the slight dry eruption of the face, sometimes erythematous, at other times furfuraceous, with some of the above lesions elsewhere. Again, there is the impetigo of the face, which persists after the crusts are removed, or the pustular condition of the scalp.

The treatment is by means of a weak sulphur ointment acting as a glandular stimulant to the defective sebaceous glands, and as the upper layers of the skin are at the same time generally thickened, salicylic acid is added to dissolve this.

Lastly, where there is much moisture, a paste is advisable as an absorbent, or if this is still intractable a paint of weak silver nitrate may be used.

In considering *warts and molluscum contagiosum* one must keep in mind that they are strongly infective, and that the latter can often be traced directly or indirectly to public baths.

Tuberculosis of the skin is too wide a subject to discuss here, but in diagnosis remember that it is of long duration, and with reference to treatment consideration should be given to the individual's health, and all cases should be kept under observation for two years after the signs of the disease are absent.

No attempt has been made in this paper to cover even the more common diseases, and the details mentioned are only to be regarded as hints culled from a fairly extensive hospital experience.

May 19th, 1913.

Landry's Paralysis caused by Salvarsan.—R. Fleischmann had a syphilitic patient who, 13 days after a second injection of salvarsan, was attacked with typical Landry's paralysis, from which he died. He believes it was caused by the salvarsan and not by the syphilis, because a Wassermann examination of the blood and spinal fluid was negative at the onset of the paralysis; there was no lymphocytosis, no infiltration of the vessels and no sign of meningitis or myelitis, as in syphilis, but there were atrophic changes of the ganglion cells, which even Benario concedes to be evidence of a toxic effect of the salvarsan.—*Journ. A.M.A.*, vol. lx.

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A CLINICAL LECTURE

ON

SOME COMMON MISTAKES IN THE TREATMENT OF THE DISEASES OF WOMEN.*

By F. J. McCANN, M.D.Edin., F.R.C.S.Eng.,
Surgeon, Samaritan Free Hospital for Women, London.

Ladies and Gentlemen,—You will remember that last session I gave a lecture † on "Common Mistakes in the Diagnosis of Diseases in Women," and in continuance of that, I propose to-night to refer to some of the mistakes in the treatment of diseases in women.

First of all, in the treatment of such a simple condition as urethral caruncle one finds how prone it is to recur. The reason of that is, that a portion of the caruncle grows from inside the urethra, and unless that portion inside is removed in addition to that which is external, you get a recurrence from that little piece; and no doubt many of you have met with patients who have had to undergo two or three operations for this condition. It is usual to treat urethral caruncle with the cautery. Now such treatment is not infrequently followed by the occurrence of stricture. I have seen at least three examples of marked urethral stricture following on the use of the cautery. For that reason I never employ the cautery in the treatment of this condition, preferring to excise the whole caruncle, and bring the mucous membrane of the urethra to the mucous membrane round the urethral orifice by fine sutures of catgut.

Another condition frequently leading to mistakes in treatment is pruritus in elderly women. Pruritus in elderly women is frequently a precursor of car-

* Delivered at the Polyclinic.

† Clinical Journal, May 15th, 1912, p. 86.

cinoma of the labium. Therefore do not continue treating a case of pruritus in an elderly woman without taking trouble to ascertain that you are not dealing with a carcinoma of the labium. Carcinomata of the labia are sometimes treated as warts. In their earlier stages certain forms appear as a wart-like growth; and only recently I had brought to me a lady who was only æt. 42 years, who had three applications of the cautery on account of a supposed warty condition on the left labium. On examining the inguinal glands on the left side I found them enlarged, and when the growth was excised not only was it cancerous, but there was found to be a cancerous deposit in the lymphatic glands.

I need hardly remind you also that gummata in this region are often treated as cancers. One patient to whom I referred in a former lecture was intending to undergo a course of radium, but was found to have a gumma excavating each labium. The whole condition cleared up under antisyphilitic treatment.

Another common condition met with in the vulva is a chronic gleet of the ducts of Bartholin. Continually one finds this is treated by vaginal injections, on the supposition that the discharge is coming from the vagina. In all cases of vaginal discharge you should make a careful examination of the ducts of Bartholin, which open external to the hymen, so as to be sure there is not a chronic gleety discharge issuing from them. And if you discover the source the correct treatment is to slit up those ducts with a canaliculus knife, so as to allow free exit to the discharge

Another condition which leads to mistakes in treatment is an intermittent discharge from an infected Bartholinian cyst. A patient will come to you and say she has had a swelling of one or other labium, which has suddenly disappeared, and that previous to its disappearance there was a free discharge. You may examine without finding anything at all; but if that patient comes again, you may be fortunate in discovering the existence of a swelling the contents of which have re-accumulated prior to a further discharge taking place. Those cases are a little puzzling, because you may examine a patient and find nothing at all, when in reality what is taking place is an intermittent discharge from an infected Bartholinian cyst.

A number of cases associated with the clinical symptom of vaginal pain lead to mistakes in treatment, and these are often due to anal fissures or hæmorrhoids, and unless the anal fissure or the hæmorrhoids be treated, the vaginal pain persists. I have known cases treated by all kinds of sedative pessaries without benefit, which were cured by treatment of anal fissure. Therefore do not forget the influence of anal and rectal troubles in causing symptoms referred to the vagina and urethra, and even the uterus itself.

Many mistakes too, occur in connection with frequency of micturition. I think you will admit that that is probably one of the great causes of mistakes in treatment, namely, not accurately gauging the origin of the frequency of micturition. In this connection I need only refer to the association of frequent micturition with commencing tuberculosis of the kidney. I know of one very serious mistake which was made in that regard, and the patient migrated to the continent, where she saw a distinguished surgeon who removed her kidney. She had been treated by having the bladder washed out when there was really nothing the matter with her bladder; her trouble was renal.

Those cases, too, are puzzling in which there is *Bacillus coli* cystitis. Many examples of frequency of micturition defy all treatment until you come to make a bacteriological examination of the urine, and in many of the cases, as you know, not only in young women, but also elderly women, you discover the *Bacillus coli*. A vaccine will cure such cases completely.

This symptom of frequency of micturition, I need hardly remind you, is sometimes associated with urêteral calculus, which is another puzzling cause, and one which you will readily understand may easily be overlooked.

We come now to a consideration of the treatment of certain affections of the vagina. One of the most frequent of these treated wrongly is, in my opinion, gonorrhœal vaginitis, the treatment of which by injections I do not regard with favour. There is no doubt that if direct treatment of gonococcal infection in the female were carried out in all cases at an early date, you would stamp out the disease quickly. At present, either the patient begins treatment herself by injections, or she is ordered to do so, and the condition is made worse in consequence. Just think for a moment how you can get at the poison in the female, compared with the facility in the male. By means of strong

carbolic or strong nitrate of silver you can swab out the vagina and the cervical canal. Thus by a few applications you can stamp out the disease.

Another potent source of error is the treatment of vaginal discharges. I have seen a patient who had had a prolonged course of iron for the purpose of curing a vaginal discharge, when what was the matter with that vagina was that it contained a foreign body. So that a profuse vaginal discharge, occurring in an otherwise healthy virgin, should suggest to you the presence of a foreign body.

In the treatment of dyspareunia one may fall into error by employing repeated dilatation of the vagina when the cause may really be male impotence. If you have dilated the vagina and satisfied yourself that the dilatation is thorough, you will do well to bear in mind the suggestion I offer as to the cause possibly lying in another direction.

Coming now to uterine conditions, one of the commonest mistakes in treatment is the employment of pessaries when the uterus is fixed in a backward position. A pessary so applied frequently causes so much pain that the patient at once demands that it shall be removed. If, on the other hand, she bears the pain caused by the pessary, that pessary is not going to do her good. Those cases, of course, are really instances of uterine adnexal inflammation, such inflammation binding the uterus down, and fixing it in the pelvis in a backward position. Therefore the treatment for such conditions is not by a pessary, but reduction of the inflammation by an anti-inflammatory course of treatment, repeated blistering externally, *i. e.* on the flanks, or painting the fornices with iodine, and the internal administration of iodide of potassium. This treatment has got to be kept up continually for at least twelve months, and should be combined with rest, and the employment of saline purgatives. For these cases, too, what is called ichthyol treatment is, in my experience, of the greatest possible benefit. I think ichthyol treatment is much too little used in this country. I was reading the opinion of a recent authority, who dismissed ichthyol by saying, "Ichthyol is dark in colour, and has a nasty smell." It is quite true that the smell is an objection, but I have had such excellent results from the employment of ichthyol that I strongly advise you to use it. The ichthyol treatment shortly consists in the employment of 10 per cent. ichthyol and glycerine tampons, put into the vagina, well up into the fornices. Secondly, the employment of ichthyol vaseline, 10 per cent., rubbed into the flanks; thirdly, the internal administration of ichthyol pills, 2 to 3 grs. three times a day.

There is another condition in which I think pessaries should not be employed, and that is where displacements are discovered in nulliparous women, displacements which cause symptoms and therefore demand treatment. Especially in virgins for the reason that the pessary stretches the vagina, and the longer it is worn, the greater is the stretching, and the greater the stretching the larger is the instrument required. Secondly, in the pessary treatment of displacements in a virgin, you introduce a septic element into the vaginal canal, and with it the risk of infection. Operations for displacements as carried out at the present time are so satisfactory and so safe that there is little doubt the correct course to adopt in such cases is to advise the patient to have an operation, instead of condemning her to pessary treatment for years, and, it may be, for life.

I must now touch upon what may be summed up as probably one of the greatest abuses in present-day gynæcology; I mean the use of the curette. The curette, in my opinion, should be abandoned in the treatment of infection of the uterus, whether that infection be gonococcal, as it frequently is, staphylococcal or streptococcal, post-abortum or post-partum, or tuberculous, because in all these conditions it tends to cause spreading of the infection. If you curette a gonococcal endometritis, you are lucky if the patient does not get double salpingitis. And sometimes that is the way the diagnosis is made. You perhaps curette, and find your patient gets double salpingitis, and then you know you are dealing with a virulent infection. I am opposed to curetting the uterus post-partum or post-abortum. If you use a curette in those conditions, you should use it as a rake—that is to say, to get away the loose pieces—but not as a scraper. There is a great deal of difference between the two. What is meant to come out of the uterus will come away easily. And if you scrape, who is to tell when to stop? You do not know when to stop. You go on scraping until you have done what is called "thorough curettage." I should like the operator to see the uterus opened up after curetting, and to note the amount of local injury. This thorough curetting which you see

done in some cases is often productive of very great harm to the patient, and I have noticed chronic uterine pain follow on the operation of curetting. I need hardly remind you that, post-abortum, you can perforate the uterus if you are not careful; therefore I advise you again to use the curette as a rake, not as a scraper. And in order to get out the fragments, the use of gauze in squares wound round long forceps to swab the interior of the uterus is a most satisfactory proceeding. You will find it excellent in post-partum infection, as well as in post-abortum cases.

Another condition in which the curette should never be used is primary sterility. You are consulted by a woman who is sterile, and who has dysmenorrhœa. The usual treatment—the treatment adopted throughout the world—is dilatation and curetting. I say in those cases dilate but do not curette. You will at once ask why? The reason is that you may just make that patient permanently sterile by the damage which you have done to the endometrium by your curetting. In another condition, dysmenorrhœa associated with scanty menstruation, never use the curette. Occasionally it has happened that the menstrual periods have never recurred after the operation, and you might one day have a brick thrown through your window by an enraged patient! Remember to avoid the curette in primary sterility, and in cases of scanty menstruation.

Always make a careful bimanual examination under an anæsthetic before you use the curette, because in the presence of salpingitis or tubal enlargement all curetting is dangerous.

Curetting is frequently used for uterine bleeding. The uterine bleeding, however, may be associated with hæmatocele, and your curetting operation may set up inflammation and infection and very serious consequences. A hæmatocele would be discovered at your examination under the anæsthetic if you had not discovered it before. Again, the similarity between the histories and the physical signs of a threatened miscarriage and tubal pregnancy are so close that it is very necessary for you to make a careful bimanual examination lest it should turn out that you are not really dealing with a threatened miscarriage but with tubal pregnancy. How frequently mistakes are made over the treatment of uterine bleeding! I have recently seen a case in which a very bad mistake was made. The mistakes are especially serious when the bleeding is due to uterine cancer. It is extraordinary how, even yet, a large number of cases of uterine cancer are overlooked. I therefore give you three very simple rules to remember: (1) All irregular uterine bleeding during menstrual life demands a careful pelvic examination. If the patient be a virgin, it should be conducted under an anæsthetic, or *per rectum* (recto-abdominal), for by the latter, even without an anæsthetic, you can feel any growth there may be connected with the cervix. In a suspicious case you would have to make a more thorough examination. (2) Profuse bleeding at the menopause should give rise to the suspicion that cancer is the cause. I do not say that all patients who have profuse bleeding at the menopause have cancer; but such is suspicious of the presence of malignant disease. (3) Uterine bleeding after the menopause should be regarded as due to cancer until the contrary has been proved. In this case which I refer to, the patient had ceased menstruating ten months, and then she started bleeding again. It was called "change of life," but it was cancer. And in that case five months' valuable time had been lost, and the growth was only just removable. A diagnosis should, of course, be made at once. If those three important rules were generally remembered, it would be the means of saving countless numbers of lives, because the results which may be obtained by a more extensive operation for the removal of uterine cancer are very much better than they were formerly; and probably there is no type of cancer so amenable to surgical treatment at the present day as uterine cancer. That leads me to formulate the following statement: *that all post-climacteric uterine activity, evidenced clinically by discharge or bleeding, requires prompt surgical treatment.* That statement includes conditions of so-called senile endometritis, which are nearly all due to cancer. And in the case of the few that are not, the best treatment is hysterectomy, because they may soon become cancerous. Those rules are so simple, and, if I may say so, so to the point, that there is no excuse for anybody missing such cases. Bleeding after the menopause means cancer in most cases. There are a few examples in which a simple polypus may exist, but they are rare, and I hardly like to mention them, because I want to impress on your minds the importance of thinking that bleeding after the menopause means that there-

is cancer; and no time should be lost in adopting surgical treatment.

Now, as to the uterine appendages. "Ovaritis," a frequent source of wealth and practice to the practitioner, is frequently something else altogether. Remember the normal ovary is very sensitive; it is especially sensitive in nervous women. Therefore do not condemn women to ovaritis because the ovary is sensitive; the probability is that they have not got it at all. Treatment is frequently directed to the ovaries when the pain is really intestinal in origin. The majority of those right and left-sided pains in women are intestinal—the cæcum and appendix on the right side, the pelvic colon on the left. I do not say the ovaries and the appendages do not cause pain; they do; but you will find frequently that the ovary is treated and condemned, and even has been removed, when the origin of the pain has been intestinal. I have seen numerous cases where patients have had both ovaries removed, but it did not cure the pain, and that, from the patient's point of view, is a failure. Again, one has found cases where the right appendages have been removed, but the pain has continued until the appendix has been taken away, the appendix having been the original seat of the trouble. At the same time, it is true that the appendix may be removed when the appendages on the right side are diseased and pain still continues. Hence it must be borne in mind that no operation for appendix trouble in the female is complete until an investigation has been made of the condition of the appendages on that side. The importance of the relationship of the appendix and cæcum to the appendages on the right side has been written about a great deal, but there is also a very important relationship between the pelvic colon on the left side and its diseases and the diseases of the appendages on that side. Many cases are seen where the trouble has originated in the bowel, and has spread from that to the appendages. There are cases in which the appendages on the left side have been blamed, and have been removed, and yet the pain still continued.

When dealing with examples of double salpingitis which come to you, you should always ask yourself the question—"With what infection am I dealing?" The infections which you probably meet with are gonococcal, streptococcal, staphylococcal, tuberculous, or infection from the appendix or pelvic colon. So that when a patient comes to you with double salpingitis, you should ascertain the nature of the infection before you can proceed to a rational treatment.

I must now say a few words about what is known, in surgical slang, as "the acute abdomen," and *à propos* of that a few mistakes which are made in the treatment thereof. In cases where there is evidence of great internal bleeding, do not give salt solution until the bleeding point has been arrested. You will ask—"What am I to do?" Keep the patient absolutely quiet, and give opium or morphia. I have no doubt you may be told this is bad advice, and that you should not give opium or morphia. But if you have diagnosed great internal bleeding, or tubal pregnancy, and you are not prepared to operate yourself, I say the best treatment is to give a dose of morphia or opium, because it quietens the circulation, and minimises that terrible fear of death which a patient in that condition experiences. And after all it was the old treatment for severe bleeding in the battle-field, and is still used. To give salt solution, whether subcutaneously or *per rectum*, when you have not arrested the bleeding point, is to court disaster.

Pedicle-twisting, a frequent complication, is often regarded and treated as a "bilious attack." If you are called to a patient who has got one of these bilious attacks, make a careful examination before you prescribe treatment; you may thus discover something which you had not thought of. In cases of acute appendix trouble, do not give the patient a purgative; if you do, you may be lucky if you do not kill her straight away, and even then you will probably make the case into one of perforation. And the same statement applies to a perforated viscus, or intestinal strangulation. There are many instances where a patient has had a bottle of medicine given her, without any examination having been made, and has gone home and died of acute strangulation. In cases of acute abdominal trouble other than great internal bleeding, the best treatment is to keep the patient quiet and put hot applications on the abdomen. The public ought to know and be taught that it is a dangerous thing to take a purgative before a diagnosis has been made. The usual treatment among the laity for pains in the abdomen is to take some purgative medicine. If that pain is due to appendix trouble, perforation will occur, and even if not, the patient will be made much

worse. If due to internal strangulation, the condition is made very much worse. I operated upon a patient who had an absolutely black bowel, and who had received ten grains of calomel. So you cannot go wrong if you use the old-fashioned hot bottle. The laity should know this, and they should not take a purgative when seized with acute abdominal pain. Let the patient lie down, place a hot-water bag on the abdomen, and send for a medical man; and that medical man must not give a purgative until he has made a correct diagnosis.

Lastly, one word of warning about a series of cases which are frequently treated badly, namely, cases of constipation in elderly women. Never continue to treat constipation in an elderly women until you have satisfied yourself that there is no growth in the pelvic colon or the upper part of the rectum. Such growths are exceedingly common in elderly women at the recto-sigmoid junction, and I may say they are very amenable to treatment; they can be excised and an end-to-end anastomosis done with as much success as most abdominal operations. It is very necessary to discover this disease in an early stage. I had this forcibly impressed upon me by a patient who had had partial obstruction for a fortnight, and was having repeated doses of purgatives. That case brought home to me the importance of giving this warning, that in all cases of constipation in elderly women be sure there is not a growth in the pelvic colon or recto-sigmoid junction before you give treatment by purgatives, or continue that treatment.

If you will inwardly digest these few suggestions you will probably not have wasted your time this afternoon.

May 26th, 1913.

A Sign of Acute Pancreatitis.—C. E. Todd has had a good many cases of acute pancreatitis under his care in the Adelaide Hospital, all sent in with the diagnosis of intestinal obstruction. He has noticed in all the cases a sign which has not received attention in the text-books—*absolute duiness in both flanks unaltered by change of position.* It is due to collection of extravasated and partially digested blood in both kidney pouches.—*Australasian Med. Gaz.*, p. 279.

A POST-GRADUATE LECTURE

ON

DISORDERS OF SPEECH AS EVIDENCE OF NERVOUS DISEASE.

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As might be expected, so complicated a function as speech is exposed to many risks of derangement. The greater part of our knowledge of its disorders, however, has been gathered in the fields of anatomy and physiology, which now provide a fairly complete record of the organic side of the subject. But its psychological affections which are associated with functional conditions such as psycho-neuroses, paranoia, etc., are as yet but little understood, Accordingly, greater confidence can be felt in interpreting the defects resulting from organic lesions, though, at the same time, some elementary knowledge of the functional causes is not wanting.

With regard to the first of these two classes it will be remembered that while the intellectual control of speech is located in a part of the cerebral cortex, the mechanism for enunciating words is regulated from the spinal bulb. Any organic lesion, therefore, affecting either of these structures is likely to interfere with the speech. Again, if the fibres between the cortex and the bulb are interrupted a corresponding result is to be expected. The same holds if, below the bulb, the peripheral nerves and muscles concerned in articulation are involved.

In diagnosing any such lesion, therefore, the primary aim is to locate it either as (1) bulbar or peripheral, (2) cortico-bulbar, or (3) cortical. The subject will be presented from these three standpoints in turn, leaving the functional disorders to be dealt with afterwards.

I. Bulbar and Peripheral Affections.

Within the spinal bulb, beneath the floor of the fourth ventricle, lie the three pairs of nuclei, facial, hypoglossal and vagal, entrusted with the management of articulation. These centres are connected

by three pairs of nerves with the muscles of the lips, the tongue and the soft palate and larynx respectively. The muscles themselves, disposed along the respiratory passages, are so contrived that, like valves and stops, they can turn the current of air to use as the motive power for generating vowels and consonants.

Thus, in the larynx, the vocal cords, regulated by the vagi, are set rapidly quivering by the motion of the air until their vibrations, echoing in the empty chambers of the throat, mouth and nose, become the vowel sounds of speech (phonation). A little farther along, the soft palate, also controlled by the vagi, is able to prevent the escape of air through the nose, thus freeing the voice of any nasal twang. Finally, the tongue and lips, assisted on occasion by the palate, help to form the consonantal sounds by imposing momentary checks, partial or complete, on the current of air. The lips, for example, by checking and releasing the current, produce the consonants *p*, *b* and *w* (if the posterior nares are closed), and also *m* (if the nares are open). The under-lip alone, when set against the teeth as a partial check, forms *f* and *v*. Again, the tongue, pressing against the teeth on the front of the palate to cause a complete check, will be responsible for *t*, *l*, *d* and *r*, when the nares are shut, and for *n* and *ng* when they are open.* If, however, the tongue imposes no more than a partial check, the sounds *sh*, *th*, *ch*, *s* and *z* are formed. Lastly, when the tongue is pressed against the back of the hard palate, the release of this complete check occasions the gutturals *g*, *k* and *y*.

Now the smooth working of this articulatory mechanism is possible, apart from any question of cortical control, only when the bulbar nuclei, the nerves and the muscles are all healthy; and yet each of these is sometimes thrown out of gear. The nuclei will almost certainly be involved, sooner or later, in any of the varieties of bulbar palsy (glosso-labio-laryngeal paralysis)—whether by an ordinary chronic bulbar paralysis, by advanced progressive muscular atrophy or amyotrophic lateral sclerosis, by thrombosis (usually syphilitic), hæmorrhage or embolism, by a tumour or by an acute infection (polio-encephalitis inferior).

* The part played by the nares is disclosed when the nose is obstructed, as by a cold in the head, when, for example, "my new ring" becomes "by dew rig."

In each of these, since none of the nuclei is likely to escape, the palsy is correspondingly extensive: though, in the more chronic cases, at any rate, the tongue goes first. The patient meets his earliest embarrassments, therefore, in consonants such as *t*, *l*, *d*, *sh*, *k*, *g* and *y*, as well as with the vowel *e*. Later, when his facial muscles are involved, the labials and labio-dentals will be interfered with, and, later still, if the soft palate begins to fail, a nasal resonance can be detected in all he says. Finally, with the paralysis of the cords he phonates with difficulty.

But when the lesion lies, not in the bulb, but in one of the nerves, the effect, of course, will not be nearly so wide-spread. Indeed, with hypoglossal paralysis the awkwardness with the linguals is never considerable, provided that only one half of the tongue is paralysed, and it may hardly be detected. Similarly with seventh-nerve palsy, the labials may lose their naturally sharp outline, but not notably. It is with vagal paralysis, however, that the most obvious effects are to be heard, since in these cases the palate or the cords are affected. If the former, a nasal twang is noticeable, especially with the consonants which require the current of air to be shut off from the nose—*p*, for example. More important still, when the recurrent fibres of the vagus are interfered with, and one or other cord is weakened or paralysed, the voice becomes strange and harsh, though articulation itself remains easy enough.

Finally, in some patients, but not many, a peculiarity of speech may be due to a primary muscular dystrophy. These cases comprise only advanced stages of facio-scapulo-humeral (Landouzy-Dejerine) paralysis, and of myasthenia gravis, in the latter of which, as the patient talks, the muscles tire so quickly as to blur his articulation for the time being.

The diagnosis of any of the above-mentioned lesions is, of course, largely a matter of routine neuro-muscular testing. After satisfying yourself as to the patient's phonation, begin by putting the several muscles through their actions. This is best done by giving the patient sequences of test-words to repeat, the following examples serving this purpose very well: For the linguals, "She stood in tears amid the alien corn"; for the labials, "Fill'd with pervading brilliance and perfume"; and for the gutturals, "A yearling . . . unconscious

of the galling yoke." To test the soft palate no word is more suitable than the monosyllable "pig," which readily betrays any undue nasal colour. The results of these tests should then be confirmed by watching the muscles as they contract. If only the lips, tongue, or palate is found with defective movements, the probability points to a peripheral nerve-lesion, especially when the signs are unilateral; but with all three affected, especially on both sides, the bulbar nuclei are surely at fault. Next, corroborative signs should be looked for, such as paralysis of the muscles of expression, fibrillary twitching of the tongue, anæsthesia of the pharynx, and, perhaps, difficulty in swallowing. Finally, the electrical reactions should be tested, because, if these are lost, the lesion is certainly no higher than the bulb.

The nature of a bulbar lesion will probably be inferred readily enough from the history of the case, and from the other symptoms that may be present. In this connection it should be remembered that a tumour in or near the bulb is likely, by increasing the intra-cranial pressure, to set up sickness and convulsions.

II. Cortico-bulbar (Subcortical) Lesions.

The upper motor neurons, or cortico-bulbar fibres, linking bulb to cortex, provide the only path for impulses from the cortical centres to the bulbar nuclei and thence to the muscles of articulation. But, being frequently injured, especially by hæmorrhage, as they crowd through the internal capsule, they are often unable to convey these impulses. In such cases the medullary nuclei, thrown more or less on their own initiative, can hardly avoid bungling the delicate movements required for clear articulation. Even with a slight apoplexy, therefore, the patient's speech is wanting in definition and speed; but if, after recovering from the hæmorrhage on one side of the brain, he suffers a repetition on the opposite side, his articulation will be so impeded as to suggest chronic bulbar paralysis. Indeed, these cases have been given the not very appropriate name of "pseudo-bulbar paralysis," though, as will be pointed out immediately, this counterfeit form is usually distinguishable without difficulty from the genuine bulbar paralysis.

Apart from capsular hæmorrhage, general paralysis makes the speech thick and blurred; and, if this disease be accepted as primarily a degeneration of the cerebral neurons, the lesion affecting the speech may be regarded as subcortical. Whether in the same group should be placed alcoholism, paralysis agitans and chorea, each of which blurs the speech, is not so sure, though, on the other hand, disseminated sclerosis with its staccato speech, and Friedreich's ataxy, when the speech is jerky and hesitating, probably represent degenerative processes in the subcortical region.

However, the diagnostic point common to all these cortico-bulbar conditions is that none of them produces atrophy or electrical changes in the muscles. Apoplexy, with its sudden onset, and, probably, hemiplegia, is usually obvious enough, though when the hæmorrhage is slight, picking out only a few fibres, the diagnosis needs care. General paralysis with its muscular tremor, Argyll-Robertson pupils and mental changes will hardly offer any real difficulty, nor disseminated sclerosis, once its nystagmus and intention tremors have made their appearance.

Even the distinction between bilateral cerebral hæmorrhage and bulbar palsy is not likely to be obscure unless the patient first comes under notice on the occasion of the second hæmorrhage and without any history of the earlier seizure. The case, however, will be identified as soon as the evidence of old-standing hemiplegia has been found, as well as by the absence of fibrillary twitchings, atrophy and electrical changes, and by the emotional state of the patient.

III. Cortical Lesions.

The bulbar centres, nerves and muscles already considered, though physiologically active even in a child at birth, are not at first under any cortical control, and though no doubt a newborn infant will cry, this is merely a bulbar reflex within the capacity even of a brainless monster. So far as the cortex is concerned, everything has yet to be learned that goes to the making of fluent speaking and writing. When an infant is acquiring his earliest powers of articulate speech, the memories of a few simple sounds suggesting specific objects and actions are stored away in its cortex, probably in a special centre. Next, this centre helps in developing under its own supervision a motor centre whose function is to acquire control over

the articulatory apparatus described above. With this assistance the original auditory centre assures for itself a means for securing the reproduction of the sound-memories in its own store; and ultimately, after long practice, the two centres succeed in establishing a delicate control over the movements of the lips, tongue and palate.

Some years later, when the child comes to learn his letters, yet another centre is established—this time in the visual cortex—for the visual memories of written and printed words. All the same, since a child learns his letters and printed words by their sounds, nothing enters the new centre except after consultation with the auditory centre. Later still, when the visual centre requires a motor writing-centre for repeating its visual word-images, the auditory centre is again interested in the new development, not only indirectly through the visual centre, but directly by establishing associative bonds between itself and the writing centre.

Of these four cortical areas, therefore, the auditory, being longest established and having taken part in developing each of the others, can claim a predominant importance. On the other hand, the visual word-centre and the writing centre, being late comers, are of less account, and, indeed, except among the educated, play no great part in the mental life of the individual.

Here it should be particularly noticed that the two sensory centres, auditory and visual, are engaged partly in receiving and interpreting incoming impressions, partly in reviving these impressions for transmission along the association fibres to other centres. A cerebral lesion, therefore, even involving the cortex alone, would put a stop to both these functions, but if it lay more deeply, picking out merely subcortical fibres, the interference would be limited to transmission only. In theory, at any rate, this is possible, but whether, in point of clinical fact, cases exist of pure cortical or pure subcortical aphasia respectively at each of the four centres, is another matter. Indeed, when it is remembered that aphasia, whatever its clinical form, is usually the result of gross cerebral diseases like thrombosis or embolism, the probability is not small of both cortex and sub-cortex being involved in a common disaster. Not only that, but with more centres than one drawing their blood-supplies from the same main cerebral vessel, an arterial obstruction can easily produce symptoms covering several varieties of aphasia; and, of course, experience shows that these are the commonest cases. On the other hand, with a small hæmorrhage, tumour or abscess, it might well be that some conducting fibres would be damaged without the cortex being involved at all. In such cases the comparatively simple results have been classified as "intercortical" aphasias; probably there are several varieties of them, two of which will be mentioned later. To sum up, therefore, it will be recognised that the elaborate investigation of cases of aphasia set out in the books is not often necessary in clinical practice, however considerable its pathological importance—sometimes because the lesion is so gross as to be localised and diagnosed without the need of unravelling the patient's tangled speech-defects, sometimes because, when a simple aphasia of conduction is caused by a small lesion, this evidence is of little use in locating the tumour (or whatever the trouble may be), for want of exact knowledge of the anatomical courses of the association tracts. The following descriptions, therefore, without going into superfluous detail, aim at rendering a general outline of the orthodox classification.

To begin with the earliest acquired faculty, that of understanding words, we have *word-deafness*. The patient hears what is said to him, but understands it not; even among his own kindred he finds himself listening to a foreign tongue; he can neither repeat what is said to him nor write it down. If, wishing to speak, he tries to revive his memories of how words sound, he uses words which do not express his thoughts. And yet, by falling back on his visual memories, he may be able, by this roundabout route, to find the correct names for the things that are put before him, and to read aloud, though only imperfectly, since the visual centre suffers for want of the help of the auditory centre. It is characteristic of word-deafness that the patient does not in the least perceive what a muddle he is making of his words.

In *word-blindness* the visual memories of written and printed words are lost, partially or wholly. The patient either fails to recognise single letters, or, even if he can spell out his letters, cannot combine them into syllables or words. He is unable, therefore, to read (alexia), and, perhaps, cannot write (agraphia), at any rate from copy. But if his

auditory memories are intact, he may quite well be able to write on the stimulus of dictation, though his manuscript, when completed, may be hieroglyphics to him. Very often word-blindness and hemiopia go together.

In *motor aphasia*, the patient, though understanding both what is said to him and what he reads, can neither speak properly of his own accord nor read aloud. It is characteristic of such a case that he is perfectly conscious of his difficulty, and will work himself into a great to-do when the words will not come off his tongue. Further, his writing is affected, but then, of course, if the case is one of apoplexy, and the right arm is paralysed, who is to say if a true agraphia lies behind the paralysis of the hand? Nevertheless, the point is worth making, that agraphia may be either motor or sensory in origin.

Finally, a word must be said of the intercortical aphasias, which, as has been mentioned, are produced when the association fibres between the centres are broken. If the interruption lies between the auditory and visual centres, the patient may recognise a thing that he sees and yet cannot give it a name; but on being told the name, he recognises it at once as correct. Conversely, when a name is spoken to him, he fails to picture the object designated; though as soon as his eyes light on the object, he connects it at once with the name he has heard. This is an instance of *intercortical sensory aphasia.*

Similarly, when the connection between the auditory and the speech centres is interrupted, the patient, though understanding all that comes in by the eyes and ears, finds his difficulty in repeating what he hears, and talks nonsense instead—a variety of *intercortical motor aphasia.*

It should be noted that these aphasias of conduction are the rule, and not the exception, and that a purely cortical aphasia is rare. Indeed, Professor Marie, emphasising the importance of the conducting tracts, has been led to deny the importance of Broca's speech-centre, a lesion of which, in his view, affects articulation rather than the intellectual control of speech; and, as is well known, he would dispute the existence of separate centres in the cortex, holding that there is but one centre for intellectual speech, namely, in the left temporo-sphenoidal lobe. However, the whole subject, just at present, is so little settled that an open mind should be kept. It will be enough to remember the broad features of the various clinical types; that they are caused by cerebral thrombosis, embolism, or hæmorrhage, by tumours and by abscesses; and that the aphasic symptoms may be a help in localising the disease.

IV. Functional Disturbances.

Apart from organic lesions, many cases of aphasia are functional. It has been claimed by Janet that every disturbance of speech which can result from organic brain disease may also occur from hysteria, and he goes so far as to express his belief that the pathology of aphasia is even better studied in the hysterical examples. Certainly these functional aphasias would appear to be commoner than is sometimes appreciated. A case of word-deafness in a man recently under my observation proved to be psycho-neurotic, and Janet records a case of hysterical motor aphasia in a girl who, whenever she attempted to speak, could only iterate a jingle of nonsense words—"petit bedable, chapetit bedable"—and who, though understanding what was said to her, was unable to read aloud. Nevertheless, hysterical aphasics have a something about them which helps to distinguish them. Like all psycho-neurotics, they bear their misfortune with an unnatural equanimity—they seem content not to be cured; whereas most organic aphasics feel their plight acutely, are annoyed by the nonsense that issues from their mouth, and make desperate efforts to control it. Then, again, the hysterical patients, notwithstanding their cerebral symptoms, seem in no way mentally dull, such as might be expected if the brain were diseased.

Another functional trouble, commoner than aphasia, is aphonia—the loss of the voice except for whispering. This condition, which implies a partial paralysis of the vocal cords, is to be known from any nuclear or vagal paralysis by its sudden onset, the absence of other organic signs, the female sex of the patient, and by the fact, ascertained on laryngoscopical examination, that only the adductor movement of the cords is paralysed. It is hardly likely to be confused with that other hysterical condition when even the whispering voice is lost and the patient becomes mute as a fish.

Yet another group of functional disorders includes the articulatory tics—spasms and twitchings of the muscles used in speaking. These tics,

common in children as well as in adults, assume almost as many forms as there are muscles. One patient, when trying to talk, catches at his breath; another makes uncouth noises in his throat; a third clicks his tongue; a fourth, trying to produce a labial, contracts his lips just when he ought to open them. Closely allied to these are the even commoner affections of stammering and stuttering, though the difficulty here lies, not, as is often supposed, in enunciating the consonants—this is done expertly enough—but in providing the blast of air to follow immediately on the heels of the consonant. That is to say, the diaphragm and intercostal muscles are not under proper control, they fail to work in with the articulatory muscles, with the result that, for example, the lips repeat some initial consonant over and over again before the belated current of air brings the vowel-sound along. So far as the origin of these articulatory tics is concerned, many of them have their roots in psychical disturbances, a circumstance which, of course, suggests the line which the investigation should follow.

May 26th, 1913.

Permanent Drainage without Drains.—Evler makes an opening by separating muscles or boring through bone, thus permitting the effusion to ooze into the subcutaneous connective tissue. This not only drains away the fluid, but has the effect of autoserotherapy. In ascites he separates the recti and draws peritoneum up over the edges of the hole thus made; the hole is only large enough to admit the tip of the finger. He usually taps the patient first. One patient thus treated two years ago has been in good health to date, and two cancer patients were immeasurably relieved. He drills a rib for pleural effusion. As the drill is removed, the fluid spurts from the hole. He has applied the method in two cases of effusion in a bursa, suturing the opened internal capsule to the external. Living tissue seems to be able to take care of a certain amount of bacteria and their toxins when the focus is drained in this way. In his three cases of effusion in a bursa the method proved so simple that none of the patients would stay in bed afterward. His only failures were in cases of effusion in the knee with deforming arthritis. The results were particularly striking in eight cases of a cystic tumour in tendon or fascia tissue on hand or foot.—*Journ. A.M.A.*, vol. lx, p. 950.

CERTAIN PERICARDIAC ADVENTITIOUS SOUNDS.*

By CAREY COOMBS, M.D., M.R.C.P.Lond.,
Assistant Physician, Bristol General Hospital.

It is a well-known clinical fact that the friction-sound of acute pericarditis is often heard first, and sometimes throughout the attack, at those areas which correspond to the contact of heart with lungs; and that in these positions the rub has a respiratory rhythm. In making a search for bedside evidence of pericarditis once active but now burnt out, marks of which are so common a feature of post-rheumatic heart disease as seen in the post-mortem room, I came upon various signs, some of which were unmistakably traceable to pericardial friction; with these this paper is not concerned. There were others, however, in which were hints of a possible pleuro-pericardial origin, as the following account of them will show.

These other sounds are divisible into two classes:

Class I: Crepitations at the Pulmonary Area of the Heart.

The more obvious type may be considered first. My notes include fifty-one examples of this adventitious sound, which consists of a series of fine crackles heard in every case during inspiration and only in one or two cases during expiration as well. In thirty-eight cases the site at which these were most clearly to be detected was the inner end of the third and fourth left interspaces; in some of these they were audible at other spots as well, and in the remaining thirteen cases they were noted, not at the pulmonic area, but at other points along the cardio-pulmonary frontier—down the left border, at the apex of the heart, in the cardio-hepatic angle, or along the sternal borders. In practically every instance this physical sign was associated with those of advanced cardiac disease of the type caused by rheumatic infection. In twenty-eight of the patients there were evidences of mitral stenosis with or without other valvular lesion; while in the remainder, with one or two

* A paper read before the Bristol Medico-Chirurgical Society.

exceptions, the signs were those of advanced ventricular dilatation and hypertrophy with mitral incompetence. The exceptions were cases of less advanced cardiac rheumatism. The average age of the patients concerned was twenty-five. The fact that these minute explosive sounds were always heard over areas where heart and lung came in contact with each other, coupled with their superficial nature, gave colour to the belief that they were due to pleuro-pericardial friction.

This hypothesis was supported by one or two other data. In several cases an attack of pericarditis was followed by the appearance of these crackles; and in one case, that of a boy, æt. 7 years, they developed in the exact spot at which the pericardial rub had been earliest and most conspicuously audible—at the inner end of the second right interspace. Added to this there was in one or two instances some association between these signs and other evidences of pleuro-pericardial friction. Finally, they are much like those crackling respiratory sounds which are sometimes heard at the base of the heart in cases of aortitis —sounds which have been ascribed to pleuro-pericardial friction.*

At the time when about a dozen examples of these phenomena had come under my notice, however, a short paper was published in the 'Lancet' (1) by Dr. C. W. Cunnington, who described the association of crepitant sounds in the third left interspace with mitral stenosis, and regarded them as intra-alveolar in origin. The ætiological factors, according to his judgment, are pressure by an enlarged left auricle and passive congestion of the lungs.

It became necessary, therefore, to decide between these two theories: whether these crackling sounds were the outcome of pleural or pleuro-pericardial friction, or whether their origin lay in congested and sticky alveoli. Of course, a complete reply to this question can be given only after an actual inspection of the parts concerned, but no opportunity of this has offered itself. Notwithstanding this lack of direct proof there are several clinical indications which point very distinctly in the direction of the alveolar theory.

First, the sounds under discussion are especially associated with the more advanced phases of rheumatic heart disease. In more than half of my patients there were signs of mitral constriction; and where these were absent there were nearly always evidences of very pronounced cardiac disease, the ventricles being much enlarged and the mitral valve grossly incompetent. In eight cases auricular breakdown had occurred, the pulse being absolutely irregular, and in several there was hæmoptysis. The cardiac conditions were therefore pre-eminently those which favour pulmonary congestion.

Second, the area over which the crackling sound was heard increased in several cases *pari passu* with increase in other evidences of hyperæmia of the lungs.

Third, the average age of the patients presenting this sign was well over twenty—that is to say, away from the age at which active inflammation of the pericardium is most apt to occur. Its incidence was in about 12 per cent. of all cases of rheumatic heart disease, and in 20 per cent. of those presenting signs of mitral stenosis.

Fourth, the fact that in nearly every instance the sounds were heard during inspiration only weighs for the alveolar and against the pericardial theory.

Fifth, as Dr. Rudolf pointed out in a letter (2) commenting on Dr. Cunnington's communication, crepitations of this type sometimes disappear after a few deep breaths; for such cases at least the pericardial hypothesis is impossible.

Lastly, similar sounds are sometimes heard at the same spot in asthmatics and persons with flat chests; here they originate in imperfectly expanded alveoli.

It may therefore be accepted that these crepitant *râles* heard at the border of the heart, and especially at the pulmonic area, in connection with rheumatic carditis and its consequences, are produced within the pulmonary alveoli, as Dr. Cunnington argued, and have nothing to do with pleuro-pericardial friction.

Two matters remain for discussion—the actual nature of the anatomical changes which give rise to these alveolar *râles*, and their practical significance. As to the anatomical question, though no direct inspection of the parts such as would make

* Observations of a case of aortic dilatation, in which sounds of this kind developed at the aortic area, have convinced me that in this instance, at any rate, the sounds were alveolar in origin, and had nothing to do with pleuro-pericardial friction. I do not know of any post-mortem data bearing on the subject.

the whole matter plain has hitherto been practicable, there are two facts of obvious importance. First, it is at the margins of the lungs that these sounds are heard, and especially at the pulmonic area, a point where in rheumatic heart disease there is a change in the relation of the cardio-pulmonary frontier to the chest-wall, as the abnormal pulsation seen at this spot in such cases testifies. Cardiograms such as I have taken in a score or so of these cases show that the pulsation is mainly or wholly ventricular, and arises from the hypertrophied and exposed conus arteriosus dexter. Probably, therefore, imperfect expansion of this part of the lung is a chief factor in their causation. The other factor is hyperæmia. These moist sounds are almost always associated with advanced obstruction or inadequacy of the mitral valve; their area increases with onset of a bronchial catarrh or administration of an expectorant, and in some it widens gradually as the condition of the heart becomes graver. Passive hyperæmia and imperfect inspiratory expansion of marginal alveoli are in all probability the forces determining the formation and retention of intra-alveolar exudation in these patients, and are thereby the factors responsible for the production of this particular type of moist sound.

Two points of practical importance still demand consideration. First, the crackles, if constantly present, and more especially if extending in area, should be regarded as evidence of persistent and increasing congestion of the lungs, and as such add a little to the gravity of the prognosis. Second, what is much more important, there is real danger lest the presence of moist sounds of this fine type in the third left space be regarded as witnessing to a tuberculous infection of the lungs, particularly when they extend upwards as high as the clavicle. Three actual examples of this error have already come under my notice, and what is more remarkable still, two papers appeared last year in a New England journal contesting the truth of the old dictum that the mitral patient does not become tuberculous, on the strength of a series of examples of coincidence of mitral disease with tuberculosis, in which the diagnosis of the latter lesion rested largely on the presence of fine alveolar *râles*. The notes of these writers' cases leave little room for doubt as to the nature of the moist sounds heard; in the majority, at any rate, they belonged to the class described in this paper. In my own cases these crepitant *râles* had nothing to do with tuberculosis; in many instances they persisted for years within a limited area and showed no tendency to spread, and, moreover, the symptoms and other signs of tuberculosis were wanting if malnutrition, cough, and occasional hæmoptysis be excepted. Nevertheless, the picture may imitate that of consumption well enough to make the differential diagnosis a matter of anxiety.

Class II: The Solitary Præcordial Click.

The second class of sound is somewhat similar. It is one which is chiefly associated in the mind of the physician with the diagnosis of tuberculosis—the solitary præcordial click, a single sharp sound like the tick of a taximeter accompanying one or other of the cardiac sounds and heard constantly at some point of the præcordium, in one case at the pulmonic area, in another at the apex, and so on. I have noted the occurrence of this type of sound in eighteen cases of rheumatic disease of the heart; in six of these it occurred at the apex only, in six at the pulmonic area only, in one at both these areas, in three cases along the left sternal margin, in one beneath the sternum, and in another over the whole præcordium. In seven the click accompanied the first sound, in seven the second, in two it accompanied both, and in two its time was not noted. If heard at the apex it is more likely to be systolic; diastolic if at the base. It was noted several times in conjunction with the respiratory crepitations described above; indeed, in one or two instances both types of sound were heard within the same small area, and within that only. It is likely, therefore, that the origin of the solitary click also is to be sought within the alveoli. Since that which it imitates is not so characteristic of tuberculosis as the volleying series of *râles*, it is less likely than the sign described above to cause confusion; but it is worth recollecting that a single click accompanying one or other or both of the cardiac sounds may be due to intra-alveolar changes other than those of tuberculosis. It is, of course, met with in chests where there is no cardiac disease, and no pulmonary disease apart from the effects of imperfect expansion of the lungs.

Summary.

Two types of adventitious sound heard in the neighbourhood of the heart, particularly if it be

the seat of rheumatic or post-rheumatic disease, are here described.

In the first class fine inspiratory crepitations are found, usually at the pulmonic area of the heart, but sometimes at other points along the cardio-pulmonary margin. These arise within flattened, hyperæmic alveoli. Their presence may give rise to an erroneous diagnosis of pulmonary tuberculosis.

In the second class a single click may be heard accompanying one or other of the cardiac sounds at any point in the præcordium. These sounds are also alveolar in origin.

REFERENCES.

(1) 'Lancet,' 1910, vol. i, p. 790
(2) *Ibid.*, 1910, vol. i, p. 1098.

May 26th, 1913.

CHLORIDE OF LIME IN SANITATION. By A. T. HOOKER. (New York: John Wiley & Sons. London: Chapman & Hall, Ltd.) Pp. 224. 12*s.* 6*d.* net.

This book deals in an interesting fashion with the merits of "chloride of lime" so called, because, in the language of chemistry, it is not a definite chemical compound, but a mixture of calcium chloride and calcium hypochlorite. Its properties seem to have been discovered early, viz. in 1799, and it came into industrial use in the following century. Like many other members of the disinfectant family, it owes its efficacy to its power of oxidation. Partly owing to its disagreeable odour, partly because it is less easy to handle, it has been displaced to some extent by the products of the destructive distillation of coal. Its domestic use has largely ceased, because while it bleached the clothes it injuriously affected the fabric. If it were only common knowledge that its use must be followed by an antichlor, such as hyposulphite of soda, its undoubted value would again be apparent. This is the secret of its continued and increasing employment in laundries, etc. The book fitly and fully enlarges on, and also justifies its powers in, water and sewage purification, street-sprinkling, flushing, and other directions.

Copious abstracts and references considerably enhance the value of the book, both tomedical officers of health and to sanitary inspectors.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from page* 48.)

(201)

Importance of Hygienic Education.

At the same time the school ought to give instruction to the child in the essentials of hygiene; if each child has a right to be taught sufficient to enable it to earn its living in after life, it surely should also be taught how to safeguard its health, without which remunerative work is almost impossible. To the argument that there is no room for this subject in the already crowded time-table of our schools, I would suggest that, after all, hygiene is of more practical utility than botany (for example), and that State-provided education should in its scheme of instruction bear in mind the ultimate good of the nation, and consider the welfare of the individual mainly in relation to the community of which he is a member. It is not enough to teach the child how to earn money for himself; education should also aim at making him a good citizen, and after all, a healthy population is one of the valuable assets of a nation.

("Some Points in relation to Tuberculosis and its Prevention," by J. Edward Squire, C.B., M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvii, p. 161.)

(202)

Expressed thought as a Stimulus.

The expression of one's thoughts challenges thought in others, and may thus lead to valuable results.

(*Ibid.*)

(203)

Age not merely a matter of Years.

It is only necessary, therefore, in confining our attention to hæmorrhagic hemiplegia, to remind you of the concise dictum of a distinguished French physician who laid down the formula "that a man is as old as his arteries."

("A Paralytic Stroke," by Seymour Taylor, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvii, p. 177.)

(204)

The Danger Zone for Hemiplegia.

A man at between fifty and sixty-five years is in the danger zone, or storm centre, of hæmorrhagic hemiplegia. His vessels have probably been the seat of decay for some years previously; but he is still in active work both of mind and body; he still is making exacting demands on his circulatory system, until some artery, weaker than the rest, suddenly gives way, and the catastrophe occurs.

(*Ibid.*)

(205)

Teetotalers not necessarily Temperate,

or

Total Abstinence no Safeguard against Hemiplegia.

On the other hand, I have seen a goodly number of hemiplegics in teetotalers. There is as much danger in the knife and fork as in the wine-glass, and total abstainers, as a class, eat more than moderate drinkers. This is the sort of daily life which many men belonging to the class wrongly termed abstainers lead, at least so far as their gastronomic exercises are concerned. A man of this class has an uncommonly good appetite, for which he is duly thankful and at the same time unduly proud of. He consumes for breakfast, ham, eggs, and perhaps a chop or some cold viand; he likes a lunch consisting of at least two "meat" courses; at dinner he indulges in good soup, followed by fish, poultry or game, butcher's meat and sweets. Probably at at least two of these meals, and sometimes between, he imbibes a large quantity of that very nasty and by no means harmless concoction called cocoa, and towards the end of the day he may pat his comfortable parietes and thank God "that he is not as other men."

A paralytic stroke will overtake that man as soon as, nay, sooner than, it will a man who does not wear a ribbon, but who has enjoyed the good things of this world in moderation and is thankful for them. If over-eating produced half the visible effects on our legs or made our speech anything like as thick as does alcohol, how many would escape the frequent attentions of the police and appearances before the magistrate's court? Intemperance, therefore, is by no means limited to alcohol.

(*Ibid.*)

(206)

"United we Stand,"

or

The Importance of Two Legs.

You must certainly regard the lower limbs as the more important, and, in addition, you must remember that we have not so much two legs as a pair of legs, one being of itself of very little use, and perhaps of no more comparative service to us than is one blade of a scissors or one blade of a forceps. Not so the arms. They are less coupled together as it were, and even one of them is of great and separate service.

(*Ibid.*)

(207)

The Advantage of real Total Abstinence to the Teetotaler.

As I have stated above, the satisfied teetotaler who eats inordinately, who drinks with much self-satisfaction large quantities of cocoa and other supposed harmless beverages, often falls an early victim to cerebral hæmorrhage. I know nothing better for these people than a twenty-four hours' fast every week. But the fast should be a real fast, and it should be observed regularly for some weeks, until the arterial tension is much reduced, and it should be regarded "as well for the body as the soul."

(*Ibid.*)

(208)

Danger of Cerebral Excitement in Arterio-sclerosis.

You know the experiment mentioned by Ferrier in his Harveian oration, by which a man, firmly fixed on a plank, is allowed to rest on a balance in equilibrium. When the man went to sleep the plank was tilted towards his feet; when he awoke the plank again assumed the horizontal position; but when he was given some exciting intelligence to read the plank was tilted towards his head, indicating the greater flow of blood to the brain and basic ganglia consequent on mental stimulus or excitement, no matter how slight. Here, then, is a physiological experiment which is of the utmost value in the treatment of arterio-sclerosis. It proves emphatically that where possible the sufferer must let his mind be freed from anxieties, and his heart from excitements.

(*Ibid.*)

(209)

"When in Doubt—Speak the Truth."

Pudd'nhead Wilson.

I have already told you the importance of confessing you do not know when you are not sure. Whenever you are not sure of a diagnosis say so, otherwise you are sure to land in trouble sooner or later.

("On the Significance of Fever in Patients from the Tropics," by Sir Patrick Manson, K.C.M.G., M.D., F.R.C.P., F.R.S., 'Clin. Journ.,' vol. xxxiii, p. 305.)

(210)

A Special Providence watches over the Gonococcus.

The usual selection of the bulbous urethra is due to the fact that this portion of the canal is extremely vascular, is richly supplied with glands and follicles and therefore appears an attractive and eminently suitable habitation for a prolonged stay on the part of the gonococcus.

("Stricture of the Urethra and its Treatment," by Cyril A. R. Nitch, M.S., F.R.C.S., 'Clin. Journ.,' vol. xxxvii, p. 183.)

MALINGERING AND FEIGNED SICKNESS. By Sir JOHN COLLIE, M.D., J.P., Medical Examiner, London County Council; Chief Medical Officer, Metropolitan Water Board; Consulting Medical Examiner to the Shipping Federation; Medical Examiner to the Sun Insurance Office, Central Insurance Company, London, Liverpool and Globe Insurance Company, and other accident offices; late Home Office Medical Referee, Workmen's Compensation Act. Assisted by ARTHUR H. SPICER, M.B., B.S.Lond., D.P.H. Figs. 44. Pp. xii + 340. London: Edward Arnold, 1913. Price 10*s.* 6*d.* net.

This book is the first attempt at anything like an adequate treatise upon malingering in the English language, and is doubly welcome at a time when the Insurance Act has become an additional incentive to malingering, already on the increase as the result of Workmen's Compensation Acts. There are twenty-six chapters, mainly devoted to the methods adopted in making medical examinations, either generally, or of the various organs and systems in cases where illnesses are feigned or imagined.

Chapter V, on functional nerve diseases, is illuminating in its differentiation and comparison of neurasthenia, hysteria, and traumatic neurasthenia. Other chapters deal with the value of X rays, and the use of electricity and its mode of application when malingering is suspected, and many useful hints are given upon the conduct of a medical witness in court, the legal position as regards libel and slander, and the medical aspect of the Workmen's Compensation Act and the Insurance Act. Another chapter is devoted to the position of legal aid societies and their fostering of malingering, while another contains a useful list of eponymic signs which a medical witness should be conversant with. Cases from the author's personal experience are freely given illustrating the various points.

The author points out the necessity in the near future for the appointment of medical referees specially qualified in this branch of practice, and the advantages to be gained thereby as seen in the few instances already adopted by some corporations, where all employees who have been on the sick-list for twenty-eight days are sent for examination and report to an independent referee. The value of such a system is seen in the decreased length of sickness, while the practitioners in attendance welcome such an arrangement, whereby they are often released from the disagreeable task of informing an employee, who is reluctant to return to work, that he is now fit. In the opinion of the author approved societies will be driven to appoint such medical referees in order to prevent abuse of their funds. As under the Insurance Act sickness benefit only becomes payable from the fourth day of illness there will be a strong temptation to prolong slight ailments.

The book is ably and clearly written, and the subject is treated in an eminently readable and interesting style, and should appeal, not only to medical men, but to all those directly or indirectly interested in the medical aspect of industrial problems.

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A CLINICAL LECTURE

ON

SALPINGO-OÖPHORITIS.*

By T. G. STEVENS, M.D., B.S., F.R.C.S., M.R.C.P.,

Obstetric Surgeon to Out-patients, St. Mary's Hospital; Gynæcologist to the Hospital for Women, Soho Square; Physician to In-patients, Queen Charlotte's Lying-in Hospital.

GENTLEMEN,—I have chosen the subject of inflammation of the tubes and ovaries, salpingo-oöphoritis, because it is an important condition, comes before us very commonly in practice, and calls for considerable judgment as to the kind of treatment which should be adopted at the different stages of the disease. Naturally, I shall not have time to go very fully into the whole subject, but I shall try and take those points which always seem to me to present difficulty in the diagnosis and treatment of the condition. I want, first of all, to point out one or two anatomical considerations as to the causation and the results of the condition in its fully developed state.

With regard to causation, we must be perfectly clear that salpingo-oöphoritis is a distinct entity; that salpingitis by itself hardly ever exists, except, of course, in the preliminary stages whilst infection is travelling from the uterus towards the pelvic peritoneum. Oöphoritis, which used to be treated of in the older text-books, does not exist by itself, and inflammation of the ovary can only occur as a result of infection. Infection obviously must come from somewhere, and it practically always comes from the generative canal, spreading up from the vagina through the uterus, through the tube to the ovary across the pelvic peritoneum. I am not now considering those cases which occur as a result of acute puerperal infection, in which the ovary may be involved with the tube and the pelvic peri-

* Delivered at the Polyclinic.

toneum, as a part of a lymphatic infection from the uterus, nor cases of tuberculous salpingo-oöphoritis where the infection may be carried by the blood-stream.

When you think of the generative system as practically a continuous series of cavities lined by epithelium, it is not difficult to realise that a catarrhal inflammation, started at the lowest point, namely the vagina, can spread through the rest of these epithelium-clad cavities to the pelvic peritoneum by direct continuity. And it is not until the peritoneum is reached that the ovary is involved in the inflammatory process. This being agreed to, it is obvious that the commonest infection must be gonococcal: it is the only common organism which has the power of spreading through the generative canal in this way by direct continuity of service. Streptococcal infections do occur, and probably as rarities, pneumococcal infections, with other organisms as secondary invading agents; but we find that infections by the streptococcus, *Bacillus coli communis* and other organisms are much more commonly the results of infection after labour than the results of infection apart from labour; whereas, of course, the gonococcus is practically always an infection which has nothing to do with labour at all. I cannot give you any figures of a reliable nature to inform you of the percentage of cases of salpingo-oöphoritis which are due to the gonococcus. This percentage has been variously stated by different observers as 40 or 80 per cent. Probably the truth lies somewhere between the two figures, but it forms by far the largest proportion of all the cases. It is not always possible to know at what date the infection has taken place which involves the tubes and ovaries. In some cases of acute gonorrhœa the tubes and ovaries become infected within six weeks of the time of infection of the cervix. I need hardly say that the cervix and the urethral orifice are the places which are first infected by gonorrhœa as a rule, because the vagina offers no suitable medium for the growth of the gonococcus. The cervix, on the other hand, forms a very suitable place for the gonococcus to grow undisturbed, in a medium which is alkaline in reaction, and therefore best suited to the growth of that particular organism. The vaginal secretions are acid, and in them the gonococcus will not grow. When I say "will not grow" I mean it will not grow so long as the vaginal secretions are normal in character. When there is a large amount of discharge set up by the inflammatory process in the cervical canal, then the normal acid secretions of the vagina are completely altered; they are swept away by the bulk and quantity of the discharge, and the reaction becomes alkaline. Then, of course, the gonococcus will grow in the vagina, just as it would anywhere else; it obtains a hold in the cervix, and from there the vagina may become secondarily infected. The uterine cavity becomes infected, and then through the uterine end of the tube the infection spreads to the lining of the tube, and finally to the pelvic peritoneum.

A point of some importance is to recognise when the pelvic peritoneum has been reached. A gonococcal infection, or any other infection of the tube itself, gives rise to very uncertain symptoms before the peritoneum is reached. We very seldom have the opportunity of diagnosing a case of pure salpingitis, because it gives rise to such very slight symptoms. But as soon as the pelvic peritoneum is reached, naturally there will be infection of the peritoneum itself, and the case will then crystallise as one of pelvic peritonitis, causing an illness more or less severe, and always ending in much the same way, by the formation of inflammatory adhesions between all the organs in the pelvic cavity. This illness, pelvic peritonitis, is practically always, in non-puerperal cases, the result of ascending infection, and its true nature is not always recognised. If you try in a number of cases in which the tubes and ovaries are involved to localise the time at which the infection of the peritoneum took place, you will find that the illness which the infection produced varied very much in severity. Sometimes it is put down to "inflammation of the bowels," whatever that may mean, sometimes it is attributed to appendicitis, and sometimes it is called ovaritis. The point is that the true nature, namely, pelvic peritonitis, is very seldom recognised; it is more often called pelvic cellulitis, which is a comparatively uncommon condition, and seldom seen now. When pelvic cellulitis does occur, it is almost invariably the result of an infection of a tear in the cervix during labour. It is never the result of gonorrhœal infection under any circumstances. But it may follow operations upon the uterus, such as curettage or splitting the cervix, if these are performed without strict asepsis. So I

will say no more now about pelvic cellulitis: its very name tells you it is an infection of the cellular tissue of the pelvis, and has nothing to do with the peritoneum at all, or with ascending infections of the uterine or tubal mucosa.

When a gonococcal or other infection has spread from the uterus to the tubes, it may show itself in two definite ways in the tube itself: either as a catarrhal inflammation of the mucous membrane or as a deeper infection of the muscle coats, giving rise in the one case to catarrhal salpingitis, and in the other case to interstitial salpingitis. These two conditions commonly exist together, as we see when we operate upon these cases. We may find, for instance, that the tube is as thick as one's thumb, with a cavity which will only admit a probe. In that case the interstitial inflammation is more marked than the catarrhal. In other cases the wall of the tube is as thin as a millimetre, and the contents are fluid, which may be watery or purulent, varying in amount, and producing the condition which we call hydrosalpinx or pyosalpinx. This is the most marked result of catarrhal inflammation, practically without any interstitial change.

Another important factor which occurs in most cases is the closure of the abdominal ostium of the tube. It is interesting to know how closure of the tube is brought about. Inflammation of the peritoneal aspect of the fimbriæ of the Fallopian tube makes the peritoneal side swell to a greater degree than the side of the mucous membrane, and the result is that the fimbriæ curl inwards, until the peritoneal surfaces of the fimbriæ come into contact, and then they naturally stick together and become cemented by the inflammatory process. After that the contents of the tube cannot escape, and the tube may then become dilated, forming a hydrosalpinx or pyosalpinx. If such a tube is cut open at the fimbriated end some radiating lines will be seen on the inner side, representing the place where the fimbriæ had turned in and become glued together, so as to close the peritoneal ostium of the tube. If the tube remains open, which is a very rare condition, it will, of course, be constantly discharging infected contents into the peritoneal cavity, but it must not be forgotten that there is seldom any cavity left to become infected after the first attack of pelvic peritonitis. This is because pelvic peritonitis fills up the pelvic cavity with a fluid exudate all round the uterus, tubes, and ovaries, which coagulates and then gradually organises, forming adhesions and sticking together the uterus, tubes, ovaries, broad ligaments, and rectum. Of course the small intestines above will become glued down as this exudate organises and shrinks. In that way the pelvic peritoneal cavity is to some extent obliterated, so that whether the tube remains closed or open, it practically will be in a closed cavity in the pelvis. From this one might expect that a patient would have one attack of pelvic peritonitis and one only. But we know that this is not strictly the case; we know that in many of these cases re-infection with some secondary organism takes place from time to time as a result of the lowered resistance of the patient to bacterial infections. I saw, not long ago, a very good case of that kind which impressed itself upon my memory. It was a girl who had a tuberculous salpingo-oöphoritis. She suffered from this for some months. It fluctuated with regard to the symptoms which it gave rise to, but on two occasions she had most acute attacks of what was thought to be typical pelvic peritonitis. It was really the initial attack of pelvic inflammation which drew attention to the fact that her tubes and ovaries were involved. The second attack occurred something like eighteen months later, and operation was then determined upon, and it was found that there was a very typical *Bacillus coli communis* infection on top of the old tuberculous salpingo-oöphoritis. It was really the *coli* infection which drew attention to the condition, gradually becoming worse, and which was primarily the result of a tuberculous lesion. This, I think, is a common way in which secondary attacks of pelvic peritonitis arise in cases in which the tubes and ovaries are already involved. In addition to hydrosalpinx or pyosalpinx, and thickening of the tube wall, there is a further condition known as tubo-ovarian cyst, or tubo-ovarian abscess, which may result from salpingo-oöphoritis. It is a common condition, and it is interesting to see how it comes about.

The tube being thickened and swollen, and having its still open fimbriated end in close proximity to the ovary, any ripe Graafian follicle which now ruptures will provide a potential cavity in the ovary, which is exposed to infection at the same time as the peritoneum, and the result is that the infection will spread at once into the interior

of the follicle and also on to the exterior of the ovary. Following this adhesions form around the tube and ovary, which to all intents and purposes now communicate with one another, so that the cavity of the tube and the potential cavity in the ovary, which is a ruptured Graafian follicle, become one continuous sac infected with some organism, generally the gonococcus. The inflammatory products in this compound sac may be watery or purulent. In the first case a tubo-ovarian cyst results, in the second case a tubo-ovarian abscess forms. The question of watery or purulent fluid in these lesions is the result of the amount of resistance of individual patients opposed to the virulence of the infecting organism. If the resistance is high, a watery fluid results and there is a hydrosalpinx or tubo-ovarian cyst; if the resistance is low, suppuration occurs and a tubo-ovarian abscess results. No doubt it is possible for a person who has a hydrosalpinx or a tubo-ovarian cyst to change those lesions for a pus-containing sac if a secondary infection occurs to which that person has very little resisting power. The actual lesion of the ovary in all these cases of infection matters very little. It does not serve any useful purpose to talk about cortical ovaritis, parenchymatous ovaritis, medullary or interstitial ovaritis, which are terms often seen in text-books. What we have to realise is that there are two definite classes of cases; one in which the surface of the ovary only is infected; the other in which the interior is infected, which can only be brought about by infection through a ruptured Graafian follicle or *viâ* the lymphatics.

Whatever the actual lesion produced by salpingo-oöphoritis, adhesions are an invariable accompaniment, and the amount, thickness and toughness of them depends very largely on the length of time during which the disease has existed, and no doubt it depends to some extent on the kind of organism which started the infection. We do not know enough to say what are the secondary infections which produce the thick, tough, almost cartilaginous adhesions which one sometimes comes across. No doubt when the amount of original fluid exudate into the pelvis is large, the adhesions it gives rise to will be thick and tough, because there is more of it to become organised. Where the initial pelvic exudate is small there is more reason to suppose that adhesions will be thin and filmy. These adhesions, of course, are salutary; they are barriers which prevent the infection spreading to the rest of the peritoneal cavity, and I may say that general peritonitis as a result of gonorrhœal infection of the tubes and ovaries is almost unknown. It is quite different, of course, with puerperal infections. In them general peritonitis is common, but you must remember that we are not dealing then with a simple case of salpingo-oöphoritis spreading by direct continuity of epithelial surface, but we are dealing with a lymphatic infection, exactly the same as the lymphatic infection which we have if we prick a finger, which may spread up to the axilla only or into the general blood-stream and cause septicæmia. There is another interesting point in connection with general peritonitis. One of its causes is said to be a ruptured pyosalpinx; but rupture of a pyosalpinx is an extraordinarily rare accident. The reason, of course, is that a pyosalpinx tucked away down at the bottom of the pelvis is in a very snug position: it is very difficult for any external cause to rupture it; and if it has existed any length of time as a rule it has very thick walls, which are re-enforced by numbers of adhesions to surrounding organs. The walls sometimes become inflamed and rotten, but even then adhesions keep forming all the time. As long as fresh peritoneum can become involved fresh adhesions will form, and so the wall always remains comparatively thick. Even if a pyosalpinx does rupture there is still a saving clause, because the contents of any pus-containing cavity in the pelvis become aseptic in process of time. No one can say how long it takes for the organisms to die out, but it is a fact that in the majority of the chronic cases of salpingo-oöphoritis with pus-formation, no organisms can be grown on the usual laboratory media, and no organisms can be demonstrated in films taken from the pus or from the wall of the abscess cavity. So it must mean that they are killed off, and that the cavities become aseptic. This has a very important bearing on operative procedures because it is very difficult to get these tubes out from the pelvis without rupturing them, meaning that the pelvic cavity is often flooded with pus during the operation, and if that pus were virulent and infective we should not expect many of these patients to survive, whereas the rule is for the majority to recover.

So much for the anatomical considerations. Now

a word about symptoms. Naturally the cases vary according to chronicity. We are all familiar with the patient who has an acute attack of pelvic peritonitis. She suffers from the usual acute abdominal pain, particularly in the lower part of the abdomen. She has rigidity below the umbilicus. You ask her to take a deep breath, and you find the epigastric region moves all right, but below the umbilicus the abdominal walls are rigid. She is extraordinarily tender and will not permit palpation, at any rate to an extent which will be of any service in locating the lesion. In a few hours, however, something in the shape of a mass can generally be felt. This is the result of the coagulation of the fluid which is thrown out around all the pelvic organs into the peritoneal cavity. If you could open up at this period you would find the pelvic cavity filled with a thin jelly-like fluid, and the intestines and all the organs will be stuck, as it were, on to this jelly-like material by plastic lymph. Later on this fluid exudate becomes harder by a process of organisation, new fibrous tissue growing into it from the peritoneal surfaces. If you watch a person who has an acute attack of salpingo-oöphoritis which has reached the peritoneum, you will find that after about a week the pain begins to subside, the tenderness is not so great, and if there has been a rise of temperature, which there nearly always is, that temperature comes down. Apart from the pain, the tenderness and the rigidity, there are very few symptoms. There may be a small amount of vomiting; there may be, and there usually is, constipation. This constipation no doubt is due to the fixation of the sigmoid and lower end of the rectum by the inflammatory mass in the pelvis, rendering it more or less incapable of contraction. If the infection is not very serious the patient recovers in about three weeks, the mass which was felt gradually subsides, and at the end of three months can hardly be felt at all. But a person who has once had an attack of that kind does not always clear up in such a satisfactorily way. Although the first attack has cleared up, the patient is liable to a second attack at any time because there is a point of lowered resistance in the pelvic cavity. Besides, there are usually active organisms in the tubes and round about the inflammatory areas, on the ovary and in the pelvic peritoneum. So the rule is that after a few months there is another attack, and probably a worse one than before. On the other hand, some cases do not clear up at all, but proceed to suppuration at once, in which case there will be prolonged rises of temperature, sweating, wasting, and a high degree of leucocytosis if a blood-count be taken. Those are cases in which the infection is more virulent from the start, and in which, in all probability, secondary infections have taken place, either from the neighbouring intestine, or by organisms which have found their way up side by side with the gonococcus. I think secondary infections in these cases of pelvic peritonitis commonly come from the bowel, because there is fixation of the pelvic colon and of the rectum, cavities in which we know organisms are constantly found, particularly the *Bacillus coli communis*, or a fæcal streptococcus, and these organisms will easily find their way through the wall of the bowel when it is fixed by an inflammatory process.

Of course, the next point of interest is the diagnosis. What we commonly see is a woman with an acute abdomen, who perhaps we have never seen before, whom we know nothing about. We do not know that she has had any source of infection. The question arises always whether the case is one which requires immediate operation, or whether it is one which can be treated from a palliative point of view. Naturally, the first thing which occurs to us is, are we dealing with a case of appendicitis? Undoubtedly in many cases there will be the greatest difficulty in distinguishing an acute appendix case from a case of acute pelvic peritonitis; because the appendix very often is partly a pelvic organ in women: it hangs over the brim of the pelvis, and inflammatory exudations around it will be partly in the pelvis. But those are exceptional cases, and there is no doubt that in the majority of appendix cases the pain is essentially localised to the right iliac region, whereas in all cases of salpingo-oöphoritis which have spread up through the tubes the lesion is bilateral invariably. Consequently there is no great localisation of pain to one side or the other. The following case, which was admitted recently to St. Mary's Hospital, serves to illustrate the difficulty of diagnosis. It was the case of a woman who really had an acute gonorrhœal salpingo-oöphoritis, with suppuration on both sides. She had, however, put a mustard leaf over the right side, in the appendix region, with the result that

her attention was drawn entirely to the pain in the skin on the right side. Nothing could be felt on bimanual examination, because the abdomen was so rigid below the umbilicus that one could not make a satisfactory examination without an anæsthetic. The pulse, temperature, and one-sided pain all indicated an acute appendix, with a localised peritonitis. An incision was therefore made just outside the right border of the rectus muscle. The appendix was found to be normal, but both tubes and ovaries contained pus, and were densely adherent to all surrounding structures. The position of the incision did not really matter, but it made an extraordinarily difficult operation to remove both tubes and ovaries through a right rectus incision. A case such as this emphasises the importance of making a careful examination of the patient under an anæsthetic before operation. Had this been done there is no doubt that both tubes and ovaries would have been felt as pelvic masses, quite unconnected with the appendix region, and the usual median incision would have made the operation easy, which otherwise was very difficult. The patient made a good recovery.

So that, apart from examination under an anæsthetic, the localisation of the pain is about the only sign we have to go upon in distinguishing between acute appendix cases and cases of acute salpingo-oöphoritis. But immediately we are able to make a satisfactory bimanual examination under an anæsthetic we find a difference in these cases. A bilaterial pelvic mass is practically never found in a primary appendix inflammation. The appendix may be involved along with the salpingo-oöphoritis, but I am fairly certain in my own mind that the primary disease is nearly always in the tube and not in the appendix. But, of course, in that case the symptoms will be rather those of pelvic peritonitis than those of appendicitis. I have seen cases in which the appendix has been removed for an acute attack of inflammation, whilst the pelvic contents, adherent suppurating tubes and ovaries, were left undisturbed, the appendix only being just involved by a surface contact with the inflamed peritoneum. A second operation was required in these cases for the removal of the tubes and ovaries. I do not believe the right ovary and tube are often deeply infected from the appendix. They may be involved in a mere surface inflammation and become adherent, but where severe lesions of the tube and ovary exist it will generally be found that the other tube and ovary are also involved, which could not be the case if it were purely appendix infection. Putting aside appendicitis, are there any other conditions which could be mistaken for acute pelvic peritonitis? Rupture of a tubal gestation or tubal abortion will cause symptoms under some circumstances which are very like those of acute salpingo-oöphoritis. The common symptoms of tubal gestation when rupture or abortion occurs are well known. There is acute pain in the lower abdomen, there may be a rise of temperature, a rapid pulse and a blood-stained loss from the uterus, whilst a history of amenorrhœa may or may not occur. A blood-stained loss from the uterus often occurs at the time of an acute attack of salpingo-oöphoritis, whilst pain and a rise of temperature and pulse always are present. On the other hand, pallor of the patient and progressive anæmia always accompany severe internal hæmorrhage, whilst the patient is often flushed in acute infective lesions. As a matter of fact, some of them will never be cleared up where the diagnosis is in doubt, because operation is not always necessary, either in salpingo-oöphoritis or in tubal abortion. We recognise that tubal abortion or tubal rupture with excessive or constant blood loss is a condition which must be operated upon at once. But practically no case of salpingo-oöphoritis in its acute stage requires operation. We prefer also not to operate in the acute stage, because we should then be dealing with a large mass of infective exudate which would be very difficult to shut off from the rest of the abdominal cavity, and it would be a very risky proceeding, whereas we know that if we wait a sufficient length of time when that exudate is walled in and organised, the organisms causing the disease may have died out, or may have lost their virulence, in which case operation will not be so hazardous. Further, it is by no means always necessary to operate at all in cases of salpingo-oöphoritis; some of them clear up completely, and the organs may be capable of exercising their functions.

In the case of pelvic infections following labour, accurate differential diagnosis is often very difficult. In the most acute cases no obvious local pelvic lesions occur and the disease is a most acute generalised septicæmia. When local inflammatory masses occur they are all practically lymphatic

infections spreading from the placental site to the pelvic peritoneum, and then secondarily involving the ovary through the lymphatics, and the tube through the lymphatics and through the abdominal ostium. An infection of the tube spreading by direct continuity of epithelium clad surfaces practically never occurs within a week or two of delivery: at this period infection always spreads by the lymphatics or by direct continuity of the cellular tissues around the cervix, cellulitis resulting. So when you see a woman two or three weeks after delivery with a pelvic mass, there is no possibility of saying that it is salpingo-oöphoritis only, but there may be a possibility of truthfully saying it is cellulitis by itself. We can judge, of course, by the typical anatomical characters of the lesion. All we can do in such a case is to decide whether an operation is necessary and leave the diagnosis until afterwards. Operation is only necessary in such cases when the patient is very ill, when the fever is persistent, and when there are signs of suppuration as shown by leucocytosis, wasting, sweating, a hectic temperature and so on.

And now as to the question of treatment. In the acute stages, when the patient is suffering from an acute pelvic mass as a result of salpingo-oöphoritis, I say at once that operation is very seldom necessary. This is because the infection is so quickly localised by adhesive peritonitis to the pelvis and because generalised peritonitis practically never occurs. Also there is always a chance that the lesion may clear up sufficiently to permit of the ovaries and tubes, or one of them at least, becoming again functionally active. There can be no doubt that there is no more danger in operating upon acute salpingo-oöphoritis than there is upon acute appendicitis. On the other hand, the risks to life in these two diseases are not in the least the same. Acute appendicitis is often at once a fatal disease; acute salpingo-oöphoritis practically never is. So we treat these cases by palliative means at first. We must relieve the patient's pain, and we can do that by means of hot fomentations to the abdomen, or hot-air applications by means of a cradle placed over the patient, with electric lamps suspended in it. It is very easy to make a home-made apparatus of this kind in a house where there is electric light, by putting two or three 16-candle-power lamps attached to a wall plug in a cradle formed of galvanised wire. This method gives more relief than hot fomentations, because a larger area can be kept hot, and the heat can be varied by keeping more or less lamps alight inside the cradle. If the pain is so great that it cannot be relieved by some such hot application, as a rule morphia will be required. But as to morphia, I warn you that it should be given in the smallest quantity which will relieve the patient, and precautions should be taken at the same time to keep the bowels open. If you give morphia in heroic doses the patient will be very grateful to you, but by so doing you will mask any symptoms which may develop. The patient is not likely to develop symptoms of a serious nature within the first week, but at the same time, as the diagnosis is sometimes in doubt, it is very wise not to give morphia in these cases if it can be avoided. Aspirin may sometimes be used as a substitute for morphia, and it will often give relief where local applications will not. There are some points which we do not always think of: for instance there may be retention of urine, and constipation is very common. If we see that the bladder is empty the patient will be all the better for it, and there is no harm in giving a saline aperient.

The next important question is, when do operations become imperative? I take it operations are imperative in the cases which suppurate early. As I have said, some of these cases suppurate at once instead of clearing up. They will require operation because the patients are extraordinarily ill, they are suffering from very great pain, and there is a risk that these suppurating sacs would possibly burst, setting free their infective contents. I believe that there is but little danger of that, as I have already said, but a patient who is in great pain, suffering from hectic temperatures, sweating, wasting, and leucocytosis, all the signs of pus-formation, must be operated upon.

In the acute stage of suppuration, in my opinion, the proper operation is performed by the vaginal route, by simple incision through the posterior fornix, and then opening and draining any pus-containing cavities which are to be found. With the patient in the lithotomy position, a large posterior speculum holding back the perinæum, an incision about two inches wide should be made immediately behind the cervix through the vaginal mucous membrane, preferably with scissors. As long as you keep behind a line drawn transversely through the os uteri, the ureters or the uterine arteries will not be injured. When the incision is made through the vaginal wall you come to the connective tissue between rectum and posterior vaginal wall, and beyond that is the peritoneum. These are cut through with scissors, and the finger is now pushed up the back of the uterus to find and perforate any pus-containing cavities. These may be in the tube, in the ovary, or amongst the adhesions in the peritoneum. If the tube is distended its walls will be friable and the finger easily perforates it. If the ovary is distended with pus, as sometimes it is in tubo-ovarian abscesses

of acute formation, its wall is soft and easily perforated, and by using the finger in this way adhesions which are walling in the disease from above are not disturbed, and therefore there are no risks of setting free pus into the general peritoneal cavity. When all the cavities which can be felt bimanually are opened, drainage must be provided. Put drainage-tubes of equal size side by side through the vaginal opening. Use two, because you can employ one for irrigating and the other for draining. It is useful under some circumstances to irrigate these cavities afterwards, but not at the time of the operation. There is no particular reason why it should not be done at the time of the operation, but there is no object in it, but afterwards you may want to wash it out, especially if the patient has slight secondary rises of temperature and perhaps the cavities are not draining well. A very large percentage of acute suppurating cases are cured by this means, but if they are not, but have secondary symptoms, like pain and pelvic tenderness, uterine congestion, dysmenorrhœa, dyspareunia, etc., the tubes and ovaries can be easily removed by an abdominal operation later. On the other hand, in the chronic cases, which come to us after salpingo-oöphoritis has existed for many months or years and in which certain very definite symptoms are complained of, such as constant backache, menstrual pain and dyspareunia, the abdominal route is the proper one to choose for curative operations. Those patients may or may not have suppurating cavities, but they all require operations. The important thing to decide is how much to do. The tubes and ovaries must be set free from their adhesions, and no more should be removed than will be absolutely essential to make the patient comfortable and well and prevent future infections. Only those organs which are hopelessly disorganised should be removed; if you can save one ovary or part of an ovary it should be done always. If you can save a tube, even though it is closed, you can open that tube and make a new ostium, and it is possible for such a patient to become pregnant afterwards. These patients may become functionally perfect although only parts of the essential organs have been saved. This is the true conservative surgery of the appendages, and it should always be carried out if possible. Fortunately these operations are very satisfactory ones, for even if there are long-standing pus collections, they are almost always sterile. Drainage is very rarely necessary as long as the pus-containing sacs can be completely removed. If drainage is deemed to be advisable because there is some doubt as to the sterility of the pus, it should be carried out by a vaginal incision and tube rather than through the abdominal incision.

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AURICULAR FIBRILLATION.*

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AURICULAR fibrillation, although unknown as a clinical phenomenon until a few years ago, already occupies a very important place among the abnormalities of the heart. When this condition is present, normal contraction of the auricle is in abeyance and is replaced by a delirious activity of the musculature of that chamber. The recognition of its existence and of its characters as met with in practice is one of the most striking results of the use of the new cardiological methods, by whose aid fresh facts bearing upon the problems of prognosis and treatment of heart disease are continually being brought to light.

The credit of having first called the attention of the profession to this condition must be ascribed to Einthoven and Wenckebach on the Continent, and to James Mackenzie and Thomas Lewis in this country. Much of Mackenzie's work, which has been the main stimulus for these studies, was carried out while engaged in busy general practice. His investigations were largely made with the polygraph, a modification of the older sphygmographs adapted for this purpose by himself.

The results of his pioneer work have been confirmed, amplified, and, in a certain degree, modified by Lewis by the use of the electrocardiograph. The writings of these authorities are in a very large measure the source of our knowledge of this subject.

Before considering abnormalities of auricular action it is necessary to review the evidence as to the normal action of the auricle. The evidence is of three kinds:

(1) *Visual.*—In many patients, especially in those whose veins are full, pulsation may be distinctly visible in the jugular vein, and it can be shown that this pulsation is transmitted to the vein from below upwards, and must, therefore, be a direct result of the cardiac systole. In most cases this pulsation, if visible, will be seen to consist of a triple wave, and comparison with the apex-beat

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will show that the first beat of the three occurs just before ventricular systole, and may therefore, perhaps, be regarded as evidence of contraction of the auricle. In other cases, notably those in which there is right-heart dilatation and in which we have reason to suspect incompetence of the tricuspid valve, the venous pulse may be synchronous with the ventricular systole as shown by the apex-beat, and, the presystolic jugular pulsation being absent, inspection of the vein affords no evidence of the action of the auricle.

(2) *Auditory.*—Normally there is no heart-sound due to auricular action, but the classical crescendo murmur and presystolic thrill of well-marked mitral stenosis are evidence of auricular contraction driving blood through a narrowed orifice into the ventricle. This observation is of historical interest, for the changes in this murmur which may occur in the course of heart-failure gave Mackenzie the idea that the action of the auricles as pumping chambers might in certain circumstances be suspended.

(3) *Instrumental.*—Two types of instrumental methods are at our disposal: first the polygraphic, secondly the electro-cardiographic. By the polygraph simultaneous records of the radial pulse and apex-beat or pressure changes in the veins of the neck or liver may be obtained. The most general combination is a record of radial and jugular pulsation, but in certain circumstances records obtained from the other sources afford more information. In normal tracings for every wave in the radial pulse there are three waves in the jugular pulse. By measurement it can be shown that the second corresponds to the carotid pulse, *i.e.* to the ventricular systole; it is called the "*c*" wave. The preceding wave is known as the "*a*" wave; in health it occurs at a constant interval of $\frac{1}{5}$ sec. before "*c*" wave and it is indisputable evidence of auricular systole. The third wave is liable to great alterations even in normal persons, dependent upon the greater or less engorgement of their veins; it may have a double summit, and is produced by the damming back of blood in the auricle and vein during the period of closure of the tricuspid valve while the ventricle is in systole; this wave is called the "*v*" wave.

Electrical methods are used to graphically record the changes in the relative electrical potential produced by the contraction of the different parts of the heart-muscle. The healthy heart gives very definite waves synchronous with the contraction of auricle and ventricle respectively, and any change in the appearances of these is suggestive of abnormality of cardiac action. Electrical instruments give valuable information which cannot be obtained from any other source, but their cost and elaborate technique renders them unsuitable for general clinical use. We have to rely mainly upon the simpler methods of investigation, forming our conclusions in the light of the fresh observations which those who are able to employ the more elaborate methods are continually revealing to us.

It is necessary next to review very briefly some of the important facts of cardiac anatomy and physiology. The heart develops as a straight muscular tube in which contractions commence at the venous end and pass as a wave of peristalsis to the arterial end. In spite of the modifications in shape which occur later this peristaltic action persists in the adult heart, and systole passes in an orderly wave of contraction from the termination of the great veins to the commencement of the aorta.

In the adult heart two types of muscle are found, differing from one another in appearance and in function. The great bulk of the organ is made up of ordinary muscle-tissue whose chief function is to contract and drive on the contents of the various chambers. The second type of muscle is of much smaller bulk, and resembles the primitive heart-cell of the developing embryo. It has many nuclei, is not markedly striated, and combines in its appearances and function some of the properties of both muscle and nerve. This tissue is called the "*primitive muscle system*" of the heart and it has two main functions—initiation of contraction and conduction of stimuli. The primitive muscle is found chiefly in certain situations. A collection or node is always to be found at the junction of the superior vena cava with the right auricle, a position corresponding to the junction of the sinus venosus of the embryo with the auricle This node was discovered by Keith and Flack and is known as the sino-auricular (*s–a*), or Keith's node. At the junction of the auricle and ventricle is a second node—the auriculo-ventricular (*a–v*), or Tawara's node. Connecting these two nodes are widely radiating strands of primitive muscle which are to be found scattered thinly through the

auricular wall. Passing down from the "*a–v*" node is a small compact strand of primitive musculature which affords the only path for the passage of impulses from the auricle to the ventricle. This is the bundle of His. It divides soon after its origin into a right and left branch. The right passes down as a compact strand on the septal wall of the right ventricle and is distributed to the musculi papillares and parietal musculature of that chamber. The left branch pierces the upper part of the interventricular septum through the "undefended space," and spreading out in a fan-shaped structure under the endocardium of the septum passes to a corresponding distribution to that of the right bundle. In some species the representative of the bundle of His is external to the wall of the auricle and ventricle; in man it is buried in the heart-wall, but it has its own blood and nerve supply and is liable to special degenerative processes.

Although all parts of this system possess the function of initiation of rhythm they do not all possess it to the same extent: those portions which are near to the venous end of the heart have a larger measure of spontaneous excitability than those lower down. Normally the inception of the cardiac contraction occurs in Keith's node, but under abnormal conditions other parts of the primitive muscle may usurp this function. Thus the ventricle is able to initiate its own rhythm should it be deprived of the usual stimuli coming down to it from the auricle, as the result of some interference with the conducting power of the bundle of His.

Auricular fibrillation can be studied in the exposed and living heart of the dog in the course of physiological experiment. Lewis thus describes the appearances seen: "The walls of the auricle stand in the diastolic positions: systole, either complete or partial, is never accomplished; the wall, as a whole, is stationary, but careful examination of the muscle reveals an extremely active condition—it appears to be alive with movement; rapid, minute and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface."

This abnormal auricular activity may be due to absence of the master-rhythm of the sino-auricular node, associated with an enhanced excitability of usually subordinate areas in the primitive muscle of the auricle. Such a combination of conditions might occur as the result of toxic irritation or inadequate nutrition of the parts involved. No constant lesion is found post-mortem in hearts which have been the subjects of this condition; in some there is fibrosis in the region of the node, but in other cases there is no definite pathological change to be seen.

Fibrillation may arise in several conditions. Rarely it is seen as a temporary phenomenon in the course of acute specific infections. The use of digitalis may sometimes induce fibrillation, particularly in the condition recently described as *auricular flutter*. Here there is a great increase in the auricular rate, which may arise to 200 or 300 beats per minute. The contractions are, however, regular, and involve the whole muscle of the auricle uniformly, thus differing from fibrillation. The ventricle usually beats at a much slower rate than the auricle, and the radial pulse-rate may be only 80. This difference between auricular and ventricular rate depends upon a partial interference with the conducting power of the bundle of His—a condition which is known as incomplete heart-block. In auricular flutter the pulse-rate is unaffected by exercise or changes in the position of the patient. The condition is treated by digitalis, and under the influence of this drug the auricles may cease to beat regularly and pass into a state of fibrillation. This change in auricular action is a stage which is to be expected in the progress towards recovery, and on withdrawal of the digitalis the auricle is likely to revert quickly to its normal and regular rate of action.

Fibrillation is seen, thirdly, in hearts which have been damaged by previous rheumatic infection, and especially in those which present deformities of the mitral valve. Such hearts form the vast majority of those which are the subject of auricular fibrillation, and they are also the most amenable to treatment.

Lastly, fibrillation is not infrequently seen in senile hearts in which degenerative changes in the myocardium have occurred as the result of impairment of the coronary circulation. These hearts are far less responsive to treatment than those of the preceding group—rheumatic group.

Fibrillation is sudden in its onset and sudden in its cessation, but it varies in its duration from a few seconds to ten or more years. Its inception may be unaccompanied by any subjective symptoms

but sudden præcordial or axillary pain or a sensation of rapid, irregular cardiac action is not uncommon. Objectively, when fibrillation commences in its most common form—in old rheumatic hearts with some signs of failure—there is an immediate increase in the severity of these signs. In all cases the pulse becomes completely irregular; this irregularity is an extremely striking feature, and is in itself very suggestive of the presence of fibrillation. The rate of the pulse is usually considerably increased, but if there is impairment of the conducting mechanism of the heart the rise in pulse-rate may not be great. If much acceleration is present this is an indication of a healthy condition of the bundle of His, and those cases in which it occurs usually react well to treatment. Coincident with the inception of the arrhythmia there is often a considerable increase in the dilatation of the right heart, and in the cyanosis and respiratory distress, the liver may become greatly enlarged and pulsation may be detected in it, œdema may occur, and the secretion of urine may be much decreased. Venous pulse-tracings reveal the complete absence of any wave attributable to auricular systole. If a presystolic murmur was previously audible it disappears and is replaced by a soft diastolic murmur. The above signs of gross cardiac failure may not be present in senile hearts; they are only seen typically in cases of rheumatic morbus cordis of some standing and in which some degree of failure has already occurred, especially in association with right-heart dilation secondary to disease of the mitral valve. The onset of the condition may occasionally be the direct result of physical injury or nervous shock. In a man whom I had under observation for several months the fibrillation commenced immediately after a fall into the river from a barge. Another patient was in hospital here recently in whom the condition immediately followed a blow on the chest from a fall against a table; in this case old-standing mitral stenosis was present.

Treatment.—This resolves itself into an observance of the general rules of treatment of heart-failure and the use of digitalis, strophanthus or squills. The patient must, of course, be confined to bed. If cyanosis and right heart dilatation are great, the abstraction of six ounces of blood by venesection or the use of leeches will often be of great help; apparently by relieving some of the distension of the heart, it paves the way for the action of the cardiac remedies. It is impossible to exaggerate the importance of rest, and both physical and mental calm should be secured. In this connection I would strongly advocate the earlier and bolder use of morphia in heart-failure. the dangers of this drug in these cases are not as great as has been supposed, and the benefit from its use is often great. A contra-indication to the use of morphia is the presence of a pleural effusion reaching to the sixth rib posteriorly, or the occurrence of nausea and vomiting. In the absence of these complications, the restlessness of heart-failure, especially if accompanied by pain, is, in my opinion, a direct indication for the use of the drug. Attention to the bowels and the modification of the diet, so as to avoid food which is indigestible or likely to give rise to flatulence, are as important as their necessity is obvious. Starvation for the first twenty-four or thirty-six hours is often beneficial, and is rarely resented by the patient. With regard to the aspiration of ascites and the puncture of œdematous legs, these measures are seldom necessary, but if a considerable pleural effusion is present and the breathing is much embarrassed, it is a good practice to aspirate the chest. Especial care is necessary to withdraw the fluid slowly and without forcible suction, owing to the danger of syncope.

The drugs which are of value are digitalis, strophanthus and squills. These have a similar action, but I shall speak in detail of digitalis only, for it is the drug in most common use, it gives eminently satisfactory results, and it is free from some of the disadvantages of the others, *e.g.* it is less nauseating than squills, and its liquid preparations are more stable than those of strophanthus. In patients who have a high blood-pressure digitalis is not contra-indicated, for careful observations have shown that it does not raise the blood-pressure in these cases as was supposed.

Digitalis has the disadvantage of being somewhat slow in action. Therefore it is well, while commencing its administration at once, to give three or four hypodermic injections of digitalin $\frac{1}{100}$ gr. or strophanthin $\frac{1}{100}$ gr. during the first thirty-six hours.

Very satisfactory results follow the use of tincture of digitalis, in doses of 20 minims, three times a day. It is best given alone in water without any other drug. It should be continued until symptoms of intolerance appear, unless the desired therapeutic effect is obtained with a smaller quantity of the drug. The unpleasant effects which may arise are nausea and frontal headache of varying severity. Should these occur, the drug must be discontinued for two days and then administered again in smaller doses. A later effect, and one which shows that the heart is fully under the influence of the drug, is "coupling" of the beats. To detect this the heart itself should be examined, for the second beat of the pair is some-

times too feeble to reach the wrist, and an examination of the radial pulse alone would fail to reveal the condition present. This coupling need not be taken as an indication for withdrawing digitalis altogether, but when it occurs the dose should be reduced. The beneficial effect of digitalis is usually heralded by a striking fall in the pulse-rate: diuresis occurs only in those cases in which œdema is present. Coincident with the fall of pulse-rate there may be some diminution in cardiac dilatation, but this is not constant and is often very slight. At the same time there is a great improvement in the subjective feelings of the patient.

The action of the drug may be either to control the fibrillation and restore the normal auricular rhythm, or it may be confined to a steadying of the ventricle by interference with the passage of the irregular impulses down the bundle of His. The first result, which is, of course, the more desirable, is hardly to be expected if the fibrillation has been of long standing: it is seen typically when the abnormality is quite recent and is occurring in an early attack of failure in a rheumatic heart. In the old fibrotic heart it is practically never obtained. By interference with conductivity, however, digitalis is able to exert a very beneficial effect upon the ventricle. Owing to the partial heart-block produced by its action the ventricle is spared some of the continuous succession of irregular stimuli which come storming down upon it from the fibrillating auricle, and is able to recuperate more satisfactorily between its beats, which are, therefore, stronger and more effective. Indeed, so useful is this effect that patients may get up, take exercise, and even return to a moderately active existence in comfort with auricles that are still fibrillating provided that the ventricle is kept steady by the continuous influence of digitalis. For this purpose small doses, *e.g.* 5 minims of the tincture twice a day, may be sufficient, but the correct amount is easily found by experiment in any given case. To long as sufficient digitalis is given to keep the ventricular rate down to 70 or 80 the patient can continue in comfort, even for years. The pulse in such a case does not become regular, but so long as it is not allowed to become rapid the patient can enjoy a comfortable and fairly active life. Such a state of affairs is often seen in elderly men in whom fibrillation has ensued upon chronic degenerative changes in the myocardium. It is also seen in rheumatic hearts where the abnormality has not been treated early and has become persistent. In early rheumatic cases there is a good prospect of complete restoration to the normal auricular rhythm, but a heart whose auricle has once been in a state of fibrillation is prone to further attacks of the condition, and each attack tends to be more easily induced and to be more resistant to treatment than its predecessors.

June 2nd, 1913.

THE SCOPE OF SALICYLATE IN THE TREATMENT OF ACUTE RHEUMATISM.*

By REGINALD MILLER, M.D.Lond., M.R.C.P.,
Physician to Out-patients, St. Mary's Hospital, and Paddington Green Children's Hospital.

GENTLEMEN,—The subject which I propose discussing before you to-day is one of considerable importance. On the one hand we have an infection, rheumatism, which is frequent, serious, and capable of showing a great variety of symptoms; on the other we have a powerful drug, capable of exercising an anti-rheumatic action. And the questions before us are these: How far can we use salicylate to control the various manifestations of rheumatic infection? What are the limits of its usefulness, and why are there these limitations?

In practice we may lay down the rule that "rheumatism with a rise of temperature, indicates rest in bed and salicylate," but this is only a rough generalisation, and to-day we want to consider the question in more detail. I shall run rapidly through the various manifestations of rheumatism and examine their response to salicylate, and briefly conclude with a summary of the action of the drug in this infection.

Tonsillitis.—Acute rheumatic tonsillitis responds well and quickly to salicylate. The chronic enlargement of the tonsils, so very common in rheumatic subjects, is little, if at all, affected by the drug.

Muscular pains.—These are quickly relieved by salicylate, especially when it is aided by the patient's being put to bed.

Arthritis.—The prompt and very successful control of this symptom is so universally acknowledged that I need not spend time on it. I would ask you to notice three points in this connection: Firstly, the drug puts an end to the flitting of the arthritis from joint to joint, which is so characteristic of rheumatism; that is to say, that the drug appears to have the power of preventing joint-lesions by cutting short the infection. Secondly, with the subsidence of the local symptoms, there is a rapid fall of temperature—again a sign that the drug cuts short the infective process. Thirdly, where there

* Delivered at the Polyclinic, May 6th, 1913.

is a large effusion into the joint, this disappears slowly and remains for a considerable time after the subsidence of the pain and fever. This last is the only limitation of the usefulness of salicylate in rheumatic arthritis.

Pyrexia.—This is a very important matter, since it is the guide to the activity of the infective process in rheumatism just as in any other infection, *e. g.* tuberculosis.

Hyperpyrexia is nowadays exceedingly rare. It is often (when it occurs) so rapidly fatal that one must imagine that salicylate at the most can only be of secondary importance to the employment of hydrotherapeutic measures.

The high fever (103–4° F.) associated with rheumatism of the adult type (joints much and heart little affected) is usually quickly controlled by salicylate, the normal being reached about the third day. If the temperature rises again after this it usually responds to an increased dose of the drug. In children, in whom the heart tends to be much affected and the joints notably spared, the fever is generally lower than in adult cases, but it is prone to be much more persistent. I shall consider this type of fever and its reaction to salicylate under the heading of cardiac rheumatism.

Anæmia.—Hardly any infection produces anæmia more constantly or more rapidly than acute rheumatism. It results from the rheumatic toxæmia. Salicylate will not cure this anæmia; the most that can be expected of it is that it shall reduce the toxæmia by putting an end to the activity of the rheumatic infection. It is noteworthy that the administration of iron will not cure this anæmia while there are still present signs of active rheumatic infection. When these have disappeared iron is of avail, and the improvement in the condition of the blood of a rheumatic patient is a very good sign that the activity of the infection is at an end. There is no truth, I think, in the statement that the administration of iron "brings back the rheumatism." The recrudescence is due, not to the giving of iron, but to the omission of the salicylate.

Nodules.—Subcutaneous rheumatic nodules last so variable a time that it is difficult to judge if salicylate hastens their absorption. Certainly they usually disappear well as the patient gets better under the influence of salicylate. But where nodules have been present for a long while, they become hard and fibrous (originally they are fibrinous), and it is difficult to suppose that salicylate can have any very successful action on such structures. The point is not one of much importance, since the nodule itself is a harmless lesion although of serious significance, and no one wants to treat rheumatic nodules as such. It has been repeatedly stated that salicylate will not prevent the development of nodules. This I do not think can be strictly the case, since the drug very obviously tends to cut short the infection in very many cases. But since we are not able, except in the very roughest way, to foretell in what cases nodules will develop, it is impossible to judge exactly whether they are prevented or not. Certainly, however, nodules may make their appearance in patients well under the influence of salicylate. But I think more emphasis has been laid on this fact than should have been done, as there is nothing peculiar in this fact. Everyone knows that one of the most characteristic features of acute rheumatism is its great tendency to show recrudescences and relapses; and my figures show that nodules do not appear any more frequently in a relapse than does arthritis. This is of significance, for arthritis is the manifestation of rheumatism most easily cured and apparently most satisfactorily prevented by salicylate.

Headache.—This symptom, which is so common in children in the early stages of rheumatism and particularly of chorea, is very quickly relieved by salicylate. It is curious to note that headache in children is very seldom the result of the administration of salicylate, whereas in adults it is one of the commonest and earliest signs of salicylism.

The rarer manifestations of rheumatic infection, acute *bursitis* and acute *nephritis*, react in my experience very satisfactorily to salicylate. Rheumatic *pleurisy* only occurs in association with pericarditis, and does not require separate mention. *Erythema nodosum* and the so-called *Peliosis rheumatica* are not, in my opinion, true rheumatic conditions at all.

I come now to the consideration of the last two important manifestations of acute rheumatism—namely, chorea and carditis. These I have left until the end, as it is in them that it is most difficult to judge of the efficacy of salicylate. I will ask you, please, to note carefully this fact—that in chorea and carditis we are dealing with damage

done by an infection in two very highly developed organs, the brain and the heart. Both of these contain tissues which are highly organised—"noble tissues," as they are sometimes called—namely, the cerebral cortex and the myocardium. Both these tissues are capable of being severely damaged by the rheumatic toxin, and histologically we find here severe degenerative changes in the cells of the cortex and heart-muscle. These changes may be so severe that they must evidently require a long time for their repair, where repair is possible. This, then, leads us to a very important clinical point, namely, that the symptoms of chorea and cardiac dilatation may persist long after the acute rheumatic infection has come to an end, being still dependent upon a damaged state of cells in the brain and heart-muscle. It is quite essential to grasp the fact that in chorea and cardiac dilatation a complete cessation of all activity of the rheumatic infection would not lead to an immediate complete cessation of all the symptoms. Time is required for the repair of the damaged tissues. Now, in salicylate we have a drug which at the most can only put an end to the activity of the rheumatic infection, and cannot mend damaged tissues. From this it follows that in chorea and rheumatic carditis we cannot expect to obtain an immediate return to health on the part of the patient. The damaged tissues must have time to repair, even when we have put an end to the active infection. Here, then, too much must not be expected of salicylate.

Chorea.—I take the consideration of chorea first, as it is the simpler. From what I have said, it will be seen that it is proper to give salicylate in cases of chorea which are running a febrile course, or which show other rheumatic manifestations, such as pains or tonsillitis. Similarly where these have recently been present it is right to give salicylate, and probably it is wise to give a course of the drug for some time in every case, bearing in mind the great tendency towards recrudescences shown by the rheumatic infection; but, under any circumstances, the drug is to be given only with a view to diminishing the activity of the infective processes, and not in the hope of effecting an immediate cure. On the other hand, where chorea is chronic, afebrile and unaccompanied by any evidences of bacterial activity, it is correctly treated, not by salicylate, but by dietetic, sedative and re-educational measures.

Heart disease.—Here, again, where the symptoms are chiefly due to an active infection of the heart, salicylate will have a beneficial action. Thus the most favourable cases are those in which the heart is affected for the first time, the brunt of the attack is being borne by the pericardium or endocardium, and there is not a very severe dilatation of the heart. In such cases the beneficial action of salicylate may be very marked. On the other hand, where the symptoms are chiefly due to severe toxic changes in the heart-muscle, salicylate must be under a great disadvantage. Thus, unfavourable cases for this treatment are those in which there have been repeated previous attacks, the temperature is low but persistent, or there is marked dilatation of the heart, or pericardial effusion. Nevertheless, in such cases the temperature may sometimes be lowered by salicylate, if freely given, subject to the patient's being able to tolerate the drug.

And here comes in an additional difficulty in the salicylate treatment of rheumatic heart disease. It is where the heart is much dilated that the drug is likely to set up vomiting. This fact has not, I think, been sufficiently realised, and it constitutes an important limitation to the use of salicylate in this particular type of rheumatic heart disease.

I have now run through the various rheumatic manifestations and indicated the ways in which each responds to treatment by salicylate. We have seen that those that are little or not at all affected are—chronic tonsillitis, effusions, chronic or afebrile chorea, and severe dilatation of the heart consequent upon marked degeneration of the myocardium; *i.e.* manifestations chiefly dependent upon parenchymatous or toxic changes rather than upon interstitial or inflammatory changes.

Summary.

I started my remarks by suggesting that the rule that "rheumatism with fever means rest and salicylate" was a a good rule in a general way. I may now close by suggesting that the value of salicylate may be understood if the following rule is remembered: that where rheumatic activity is responsible for the symptoms salicylate works well, but where the symptoms depend upon toxic degenerative changes (as in the tissues of the brain and myocardium) the drug is at a disadvantage. It diminishes rheumatic activity but is powerless before the results of that activity.

June 2nd, 1913.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from page* 128.)

(211)

The Infallibility of the Profession.

I have come across, before now, cases where the only evidence of any injury was the statement of the patient: "The doctor says I must have had a blow," and that, in the interested party's opinion, was sufficient to make a blow an antecedent fact—an instance of touching confidence in the omniscience of the profession not always shown us.

("Surgical Cases modified by Insurance," by G. R. Turner, F.R.C.S., 'Clin. Journ.,' vol. xxxvii, p. 241.)

(212)

Put Yourself in his Place.

I take it that, however we may professionally regard the case of an unfortunate workman who has met with an accident or is ill, we all cannot help sympathising very much with the poor man whose ordinary life is, perhaps, one long toil and labour, trying to find bread for a number of youthful mouths which he has improvidently introduced into this world.

(*Ibid.*)

(213)

If no Organic Disease, what then?

A friend of his and a late President of this Society told him the story of an American lady who consulted him. He examined her with his usual care and thoroughness from head to foot, investigating every organ, and finally said, "I am very pleased to say, madam, that I find you to suffer from no organic disease whatever." "Well, then, doctor," she replied, "all I can say is I guess I don't function well."

(Discussion on "Dilatation of the Heart and Heart Strain," Sir Douglas Powell, 'Clin. Journ.,' vol. xxxvii, p. 264.)

(214)

Sore Eyelids,

or

The Value of Good Looks.

If one may judge from the advertisements that appear within the covers of the popular magazines of the day, it would seem that there is scarcely anything that is more highly valued than an agreeable personal appearance. And it is not a matter for wonder that this should be so; it is true that we have proverbs that tell us, "handsome is that handsome does," yet there is always an unconscious bias in the mind of even the most ethically disposed of moderns in favour of a pleasing exterior. A proper jewel should have a fair casket. Ancient literature abounds in quaint narratives telling how the fate of families and tribes turned on these matters of appearance, and I do not doubt that you are all familiar with the very human story of the courtship and matrimonial affairs of that primitive Hebrew chieftain named Jacob, and how the ancient writings suggest his disappointment at the manner in which the cunning Laban foisted upon him the elder and plainer of his two daughters by the note that "Leah was tender eyed." Leah had sore eyelids, and Jacob would rather have had the comelier Rachel.

("Sore Eyelids," by N. Bishop Harman, M.A., M.B.Cantab., F.R.C.S.Eng., 'Clin. Journ.,' vol. xxxiii, p. 232.)

(215)

Heart Symptoms dependent upon Stomach Disorder.

His (the speaker's) father used to say that if a person came complaining of his heart one should examine the stomach first.

(Dr. Walter Broadbent in a discussion on "Dilatation of the Heart and Heart-Strain," 'Clin. Journ.,' vol. xxxvii, p. 283.)

(216)

In searching for the Mote be careful not to overlook the Beam.

Commenting on a case of lateral curvature of the spine in a robust and healthy man, in which a course of Swedish exercises had been advised and sedulously carried out for two months, and this failing, the trouble had been ascribed to constipa-

tion, for which a course of medicinal treatment had been prescribed, with the reservation that if this failed, it might become desirable to consider the question of a serious operation, but in which on measuring the lower limbs, it was found that the one on the side towards which the convexity pointed was over half an inch shorter than the other, and that all signs of the spinal bend disappeared on placing a book of the required thickness under the foot of the shorter limb, Sir William Bennett says: "This case is mainly interesting because it affords me an opportunity of showing how easy it may be in searching for an uncommon cause to entirely overlook the obvious."

("With Sir William Bennett at the Seamen's Hospital, Greenwich," 'Clin. Journ.,' vol. xxxvii, p. 289.)

(217)

The Unpardonable Sin in Diagnosis.

The most expert and the most experienced must make mistakes in diagnosis; there is only one unpardonable sin, and that is to make a mistake as the result of careless or inadequate examination or from having failed to make any examination at all.

("When to Examine and what to look for," by Arther E. Giles, M.D., B.Sc., F.R.C.S., 'Clin. Journ.,' vol. xxxvii, p. 337.)

(218)

The Danger of Over-refinement in Diagnosis.

When the abdomen is open, and not before, is the time to consider varieties of intussusception. Then it is that the surgeon must decide whether he has to deal with the common ileo-cæcal, enteric or colic; they are all amenable to the same treatment, and merely differ in position, or, in the case of the ileo-colic, require some little extra attention in order to be certain of their complete reduction. But any difficulty encountered does not justify waste of time before operation in order to attain certainty, which, indeed, is almost impossible with an intact abdomen. If this is only thoroughly realised, much of the medical man's perplexity in these cases will disappear. Time taken up in discussing these points before operation is simply wasted. And the same is the case with regard to possible tuberculous bands, the chance of finding an internal hernia, etc.; the distinction is easily and rapidly made when the abdomen is once open; it may be impossible before. Of course, the surgeon must be competent to deal with whatever he may find.

("Acute Intussusception," by E. Stanmore Bishop, F.S.C.S.Eng. 'Clin. Journ.,' vol. xxxvii, p. 408.)

(219)

The Wassermann Reaction,

or

Laboratory Investigation an aid to, but not a substitute for, Clinical Diagnosis.

A Wassermann reaction has been done and is positive. As regards this reaction, however, about which so much has been heard recently, I would remind you that it is met with in other conditions besides syphilis, so that we must not attach to it more importance than it deserves.

The tendency of the present day is to give undue prominence to laboratory research. No one will deny the enormous help which is now given us by bacteriological investigation, but it ought to be regarded as an addendum to, and not a substitute for, clinical evidence. We all remember how the Widal reaction in enteric fever came almost to be regarded as the diagnostic criterion of the disease, until it was ascertained that it was not pathognomonic, but occurred in other conditions with which enteric fever had nothing to do. It is probable that the Wassermann test will by-and-bye, in the same way as the Widal test, be regarded as useful confirmatory evidence in doubtful cases, and no more.

("With Dr. Guthrie Rankin at the Seamen's Hospital, Greenwich," 'Clin. Journ.,' vol. xxxvii, p. 414.)

Danger from Neo-salvarsan in the Lymphatic Diathesis.—One patient was a woman of 50, the other a robust boy of 12, at the second day of scarlet fever. The positive Wassermann reaction in the first case suggested a probable syphilitic origin for the recurring liver affection. Death followed in both cases so soon after the intra-venous infusion of neo-salvarsan that a causal connection seemed beyond question. Necropsy in both cases revealed the status thymo-lymphaticus in a pronounced form. The cases teach the necessity for caution in giving salvarsan or neo-salvarsan when there is reason to suspect this condition. At necropsy after salvarsan fatalities it should be sought with special care. As exophthalmic goitre is so often accompanied by persistent thymus, it is unsafe to give salvarsan in this affection, Rindfleisch insists.—*Journ. A.M.A.*, vol. lx, p. 1402.

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THE NON-OPERATIVE TREATMENT OF MALIGNANT DISEASE.*

By T. J. HORDER, M.D., F.R.C.P.,

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THE very fact that we are met here this evening to discuss the treatment of inoperable cancer makes it clear that we lack the essential thing—the knowledge how to cure. If we had this we should have no time to spend on discussions. Yet it should not be thought, on the other hand, that, not possessing this knowledge, our talk must necessarily be futile. One insistent fact alone makes it imperative that we should from time to time consider, and consider even minutely, the means that are at our disposal for the relief of cancer—it is, that the patients are daily clamouring at our door. By free and critical discussion we may hope to arrive at a programme of treatment which shall ensure the best possible relief for the present sufferer, and also yield some hope of cure for the disease in future.

It is to be noted that the title of our discussion concedes the principle that, whatever may be the best method of radical treatment of cancer in time to come, to-day it is by operative removal whenever possible. I doubt if there is anyone here who does not freely subscribe to that principle. To-day is with the surgeon. If this is conceded, it follows that we must not only submit every operable case at the earliest possible moment to the knife, but we must consider, at certain stages in our treatment, whether a case previously non-operable has not now become operable. I take the word "operable" to include any of the means by which total removal of a cancer is undertaken, whether

* Read at the opening of a discussion at the Royal Society of Medicine.

by the knife, by the electric cautery, or by less favoured methods. Thus are we hedged about by present necessities; we cannot try any experimental form of cure upon early cases of cancer, because early cases are amenable to surgical interference, and we must not continue a form of inoperable treatment past the point at which operation becomes possible. These limitations are very severe. When we remember how difficult it is to get response to our measures of specific therapy in infective processes that are in an advanced stage, and when we are told that the experimental methods of curing mouse cancer usually fail if the tumour is more than a small percentage of the total bulk of the mouse, we realise the almost insurmountable problem underlying the therapeutics of cancer. But the position must be accepted all the same, and we must make the best of it.

The therapeutics of cancer may be considered to fall under two headings:

(I) Preventive and curative efforts.

(II) Palliative efforts.

These are, however, not distinct from each other, for some of the efforts at cure relieve the patient's pain and discomfort and prolong his life. On the other hand, curative efforts are sometimes productive of pain and discomfort, and the use of the particular remedies that have this effect should always be considered in terms of the chances of cure *in the particular patient concerned.* A few of the efforts at cure are also used as preventive measures, especially as preventive measures against "recurrence" of removed cancers.

Curative Efforts

may be subdivided according as they are *rational* or *empirical.* (1) Rational efforts may be classified according to the central fact or idea underlying them. Thus we have four of these central ideas, as follows:

(A) *The idea that cancer is due to errors in tissue metabolism capable of correction by a diet specially chosen.*—At the Cancer Hospital recently I was associated with Dr. Haig in observing the effect of a purin-free diet upon a selected number of cases of inoperable cancer. The result must be taken as equivocal, because of the great difficulty in persuading patients to give the diet a fair trial. The rationale of such a diet would need to be very convincing to justify its continuance in a wasting and depressing disease, and when faced with further loss of flesh and depression directly attributable to the diet itself. None of the arguments hitherto advanced has seemed very convincing. Even if it were true that cancer developed more readily in persons who ingested purins, this would surely only be a predisposing cause; we should be still far from a cure by merely cutting out a predisposing factor. And to cut it out with hope of success would seem to require that we start a generation back, if not still further. Almost at the same time that I had arranged to assist Dr. Haig in his experiment I was approached by a physician equally experienced in dietetics, who thought a promising diet for cancer patients was the Salisbury diet, of lean beef and hot water, and wished it made the subject of trial. The very fact that two such extremely opposite forms of diet were advocated as cures for the same disease goes very far to check enthusiasm in regard to either of them. Packard, of Boston, considers cancer to be due to the demineralisation of foods, accusing white flour and peeled potatoes as the cause of the disease. Even so, it should be a simple matter to satisfy this observer's desideratum by a full allowance of fresh fruit, by giving wholemeal bread, and by cooking potatoes in their skins. Such considerations leave us still cold. A somewhat more convincing idea, also on the lines of metabolic action, is one that Dr. Monckton Copeman will deal with immediately, and with more authority than I can claim. Dr. Copeman's hypothesis is to the effect that the cancer-cell might undergo retrogressive changes if the tissues were denied that principle in food the absence of which leads to scurvy. The diet for a cancerous patient would in this way consist of foods that had been autoclaved, as it is found that such treatment destroys this active principle. I have arranged with Dr. Copeman to give such a diet a trial at the Cancer Hospital.

(B) *That specific immunity to cancer can be established in a manner analogous to infective processes.*—It is hoped that either by the use of an anti-serum or of a vaccine the patient may acquire passive or active immunity to the disease. The procedures follow very closely upon those adopted for microbic infections. The excised tumour is macerated, extracted by suitable solvents, and the filtrate is used for hypodermic injection, either into an animal for the production of a serum or into the patient as a

vaccine. It is obvious that the living cancer-cell cannot itself be injected into the human body, so that it is necessary to use a clear filtrate or the solution of a powdered extract. But that these are capable of inducing cytolytic substances on injection, and that the intact cell is not necessary for the purpose, is rendered possible by analogy with immunisation experiments against other cell structures. The method of active immunisation is supported by the experiments on mice, which prove that the animal's resistance to cancer grafts is raised by the injection of embryonic epithelium. Despite a large number of trials, however, little or no success has attended either of these immunity methods in human cancer. This is perhaps the most appropriate place to mention the work of the Grünbaums, who found that the injection of anti-venom (horse) serum effected the regression of inoculated tumours in rats. Their experiments were suggested by the observation that these animals could not retain their susceptibility to cancer grafts, and at the same time be immunised against cobra venom.

(c) *That radio-activity can lead to retrogressive changes in the cancer-cell.*—Of all the modern methods of dealing with inoperable cancer, the two that have most success to place to their credit are undoubtedly X rays and radium. They are the most helpful measures in dealing with local recurrences and for application to certain parts that are surgically inaccessible. The choice, as between the two methods, is to be determined largely by the nature and situation of the growth. At the Cancer Hospital we very frequently employ both methods alternately in the same patient, on the principle of combined treatments, to which I shall refer more fully later. Valuable though the application of these rays and emanations are, the limits of their use at present seem all too woefully narrow. A *sine quâ non* for radium would seem to be that the growth is on the surface of the body or near to the entrance or exit of the alimentary canal, situations that admit of actual contact by the salt. For Röntgen therapy some greater degree of penetration is possible, and there is hope in the reflection that the experts have not yet exhausted their means of getting still deeper effects by careful choice of tubes and by filtration. Concurrent changes produced in the cancer tissue by some general treatment, as by the use of metals injected intravenously, may quite conceivably supplement the destructive effect of radio-active measures—another effort at combined methods of treatment. But there is one consideration that appeals in disappointing fashion in respect of both these modes of treatment of cancer, and which makes it doubtful if we can expect from them much more than we have already got. This consideration is the great range of their therapeutic use. The very fact that they are of service, and of very great service, in other diseases seems to impose a limit upon any very selective efficacy in dealing with the cancer-cell. That X-ray applications can cause a tuberculous gland to disappear, and a lymph-adenomatous one also, and can reduce the size of a leukæmic spleen enormously, seems to argue against that power of specialised effect which we feel essential in a means of cure of so specific a disease as cancer. With radium the comment seems more pertinent still, for it appears day by day more apparent that the action of radium is that of an elaborate caustic upon cancer tissue. But until we know more about the actual way in which these measures produce their effects it behoves us to keep an open mind as to their future possibilities. Neither of them is likely to be *the* cure, be its developments what they may; yet either of them may act as the key that unlocks the door to still more specific treatment. There are reasons for thinking that X-rays produce some at least of their effects by general metabolic changes rather than by local alterations in the cancer-cell: that is, they stimulate the general resistance. I think I am correct in saying that marked resorption of recurrent mammary cancer has been observed in patients who have received not local but general irradiation. If the main effect is produced in this way it is easy to understand why the rays should lack specific features for one disease. It has recently been suggested that radium acts by chemical analysis within the cancer tissue, as by liberating choline from lecithin; the choline is then thought to act in a toxic manner upon the cancer-cell. The notion has led to the employment of choline directly, by injection, but I have had no personal experience of this method of treatment.

Dr. Lewis Jones is of opinion that the good results of radio-active measures are probably produced by stimulation of the healthy tissues rather

than by destruction of the cancer-cell—in the case of carcinoma, at any rate. I incline to agree with him. Direct stimulation of the adjacent normal tissues and indirect stimulation of the body as a whole would be, perhaps, a poor exchange for the present hoped-for destruction of cancer-cells by their immediate bombardment; but if observations point to giving this exchange a good trial they should be followed. Dr. Knox's experience of the great value of raising the general resistance and of improving the hæmoglobin content of the blood seems to confirm Dr. Jones's views, though I gather that Dr. Knox still holds out hope of actual cancer-cell necrosis as a direct result of radio-active measures. Dr. Finzi certainly holds out this hope, and his enthusiasm is welcome accordingly.

(D) *That the cancer-cell is more readily destroyed by certain toxic agents than are the cells of the hosts.*—This is the principle underlying the great majority of the efforts made at curing cancer. We cling to the fact that the cancer-cell in its hasty nutrition and its disordered growth is a less stable thing than the cell which it is replacing. It should, therefore, be more readily destroyed. The natural history of the cancer-cell supports this principle, and its inherent tendency to autolysis within the tumour suggests the advisability of trying, by all legitimate means—legitimate, that is, with reference to the tissues of the host—to precipitate this destruction. The various toxic agents that have been used to compass this desire can be divided into two groups: *microbic toxins*, with which we may conveniently include diastasic ferments; and *drugs*, chiefly preparations of the heavy metals. If we are compelled to regard the chief and ultimate action of radium and of X rays as of a chemical nature, we must include these measures also under this principle, but it has been more convenient, in the present state of our knowledge, to treat of them separately.

(i) *Microbic toxins and ferments.*—Into this class of remedies come Coley's fluid, *Micrococcus neoformans*, and "antimeristem." The observed disappearance of sarcomata during an attack of erysipelas was the pioneer observation which led to treatment along these lines. My own experience of Coley's fluid, using both the market virus and material specially made, has been disappointing at the Cancer Hospital and elsewhere. And considering the fact that this method has been in use now for more than twenty years, and has during this period had very thorough trial, the infrequency with which the treatment is embarked upon to-day is proof of the lack of confidence in it. At the Cancer Hospital, some time ago, I endeavoured to raise the general resistance against streptococcal endotoxin by a preliminary course of vaccine, hoping to prevent some of the severe after-effects of the injections of Coley's fluid. This did actually occur in two cases treated, but only when the doses of the fluid were in the initial small quantities. It would seem that the desirable effects upon the growth are in direct proportion to the degree of general toxicity produced, and that without the general effect the local effect cannot be obtained. The pain and discomfort endured by the patient create, in England at all events, an almost overwhelming objection to the treatment. It is possible that, in conjunction with other methods, such as X rays and arsenic, doses of lesser size than those producing such acute suffering may be helpful. As I never expected the so-called *Micrococcus neoformans* to do anything which *Staphylococcus epidermidis albus*, of which it is a variant, could not do, I was not disappointed to find, in a series of cases treated at the Cancer Hospital in conjunction with Dr. Paine, that the only good effects were attributable to the operation of a staphylococcal vaccine. I have again and again seen undoubtedly good palliative effects from the use of bacterial vaccines in cancer. The micro-organism most commonly found in, or on the surface of, malignant growths is one or other variety of *Staphylococcus albus*. A course of staphylococcal vaccine will often relieve pain and swelling, assist in cleaning up a discharge, and reduce temperature. But more than this does not seem to occur. I think there is now fairly universal opinion that the limit of action of *Micrococcus neoformans* inoculation is that of controlling secondary staphylococcal infection of cancer, and that curative effect of the treatment is absent. Of "antimeristem," consisting of a vaccine prepared from a *mucor*, I have had no personal experience, but I cannot find definite evidence that it is of any value. *Diastasic ferments* have been largely used, on the basis of some observations tending to show that the antitryptic power of the serum is low in cancer patients. No single

method of treatment for inoperable cancer has received a more thorough trial, possibly excepting Coley's fluid, than has the trypsin treatment. Though introduced with much blare of trumpets, and boomed with abundant assurance, it has now sunk almost into the limbo of those many forgotten efforts which lie strewn along the path of cancer therapy. It sought to do too much, and thereby failed at first to evoke even a proper measure of trial; we were warned against a cure being too rapid, and were taught how to salvage the products of cancer destruction by the additional use of amylopsin. Alas! would we could believe that a patient might be killed by excessive zeal in hastening the necrosis of the hostile tissue! Imagine such a thing for one instant; that the only problem left was that of adjusting our remedy so that the cure should be safely slow instead of fatally rapid! Allied to the ferments, perhaps, are certain substances formed in exudates or transudates occurring intercurrently in cancer patients. It has been noted that cancer, on rare occasions, resorbs coincidently with the resorption of such exudates. This observation led to the treatment of cancer cases by injection of the exudate in increasing quantities. One or two partially successful cases have been reported, the most convincing being one in which the patient was so treated with her own exudate. Two years ago, with the help of Dr. Sheffield Neave, I treated six patients in this way with ascitic fluid removed from a woman with metastatic peritoneal deposits secondary to uterine cancer; but we failed to demonstrate any value in the method.

(ii) *Drugs proper.*—The second group of toxic agents used to destroy cancer-cells include chiefly the heavy metals and their compounds. The brilliant work of Ehrlich and his colleagues in connection with the spirochæte of syphilis has breathed fresh life into the flagging body of chemical therapeutics, and has given a great stimulus to research in cancer treatment. A large amount of material provided by many thousands of mice successfully grafted with cancer has been available for the purpose of such research. The highly selective action of various substances upon particular cells had led to the trial of many products specially synthesised by the chemist for the purpose. Wassermann and others have worked specially with the metals of the sulphur group, and have definitely proved that it is possible to cause the complete resorption of mouse cancer of a certain size by repeated intra-venous injection of such a compound as selenium-eosin. Eosin was chosen as part of the metal compound on account of its intensely penetrating character. The demonstration that the cancer-cell can be picked out for saturation and ultimate destruction, to the exclusion of the cells of its host, by such means as these, constitutes one of the most significant and encouraging discoveries of recent months. Whether such compounds have any similar effects in human cancer remains to be seen. The choice of the actual compound used is, from analogy with the arsenical preparations employed in the treatment of syphilis, of great importance, and the most efficient preparation—one giving highly toxic effects upon the cancer-cell, and relatively low toxic action upon the healthy tissues—may require much exhaustive experiment for its elaboration. Other observers have recorded good results in mouse cancer from the use of tellurium, and from selenium given by the mouth. Copper is also on trial. The silicates have had very extensive trial, in the form of simple sodium and potassium salts given by the mouth, at the hands of Zeller and others; and even Czerny is undecided as to whether there is not some curative effect observable from long-continued use of these preparations.

(2) Of purely *empirical* measures I propose to say nothing; their very name is legion. The 'Archives' of the Imperial Cancer Research, judging from Dr. Bashford's historical essay, and of the Cancer Hospital, judging from personal knowledge, are bursting with letters from all over the world, telling of countless "cures," secret and public. Sacks of leaves from the Antipodes cumber the shelves of our dispensary at Fulham. The violet leaves, whose reputation lately left its birthplace in Devonshire for a wider sphere, appeared to have again retired to the far western country. In respect of all such remedies it must always be remembered that to disregard the fundamental criterion is but to confuse the issue, and delay our progress—I refer, of course, to the proof of the diagnosis. Absence of histological evidence, however brilliant the result of treatment may appear, should always call for the exercise of forbearance, and a decision to remain silent. That lump at the pylorus in a wasting patient,

not all the opinions of the wisest of us are more than speculation; for a chronic ulcer may produce both signs and symptoms that deceive the very elect, and may yet get well. And so for the mediastinal mass, and for the ragged tongue, so long as there are alternative pathological processes which are capable of spontaneous recovery, with or without the assistance of rest and general treatment, the onus (and a very sacred onus) is with the champion of a cancer cure to make the proof of malignancy complete.

Palliative Efforts.

I have endeavoured in my remarks to avoid purely theoretical considerations; a few have, however, been unavoidable. The practitioner in charge of a case of inoperable cancer is only helped directly by what is practical. There are really very few cases in which there is nothing to do except negotiate euthanasia, though in the vast majority of patients this stage of the treatment arrives sooner or later. Whether a definite effort should be made in a curative direction depends much upon the nature and position of the growth, and the general state of the patient. As already indicated, it is desirable to choose those means of so-called curative treatment which carry with them relief of distressing symptoms. *The most satisfactory line to adopt in most cases is a combination of several different measures.* Not in a state of panic, but with deliberate choice of a programme. Thus, faced with an inoperable sarcoma, it might be good to lead off with an alternation of X rays and radium, and, as an intercurrent measure, give two or three doses of salvarsan intravenously at weekly intervals. In dealing with one of the most common of all types of case, that of a woman who has had a breast removed and has developed recurrence that is inoperable, X rays and radium may again be employed; vaccines should be given if there is ulceration and evidence of secondary infection; the diet may well be regulated on the basis already indicated and general tonics employed. The question is sometimes raised whether any preventive treatment should be employed—as by radio-activity—after the operative removal of a cancer. Some Continental authorities advise that the scar be treated in this way for some weeks following the operation. A few go as far as to advise keeping the wound open for a time and subjecting it to X-ray applications. This does not appear to have met with much support in England.

Partial removal of fungating or ulcerating growths may be included amongst methods of inoperable treatment, since we have defined "operation," for the purpose of this discussion, as complete removal. Such partial removal is sometimes of very considerable service, and the resultant improvement in the patient's condition may be most marked. It allows of the removal of obstructions—*e.g.* in the mouth—and of a cleaner and less septic surface. The good effect upon the mental state is also sometimes marked.

In the *relief of pain* it must, first of all, be carefully determined what the immediate cause of the pain is—whether gross pressure or infiltration, or secondary inflammation. Aspirin and other coal-tar products will often relieve pain without the use of opium. The relief of pain due to infiltration and pressure, by the use of X rays, is often most marked, and very severe neuralgias may sometimes be assuaged by this means. The customary antiseptic and analgesic lotions must be applied for discharging surfaces, and the use of a penetrating vehicle, such as acetone, is often very useful, especially in vaginal and uterine cases. Sanitas, by virtue of its peroxide, is a most useful wash and deodorant. The field of mental therapy must not be overlooked. But here it is very difficult to offer advice. The personality of the practitioner and the philosophy of the patient are the medicines that are available. Two lessons we all have probably learnt: that not every patient who "wants to know the truth" is prepared to hear it, sometimes he least who asks most boldly; and that self-deception is not seldom a valuable analgesic well worth preserving.

Conclusion.

I will not stay to labour the obvious fact that in the matter of curing cancer our efforts have scarcely even begun to receive reward. The sun has not yet risen—the proper metaphor is less than this even—there is, as yet, no glimmering of the dawn. An occasional meteor there is, a flash in the darkness, causing the foolish to cry—"Lo! here is the day!" But that same instant it is gone and the place thereof knows it no more. In the absence of facts it is always easy and tempting to be sententious, and no subject so leads to words that are rich in judicious observations, but poor in everything

else than words, as does the problem of cancer. All the same, it is imperative that we should keep our attitude right, and the right attitude I take to be divine discontent with the present, patient toil, free exchange of views and results, and an open mind. Such an attitude will assuredly lead us to knowledge at last. Every field of human endeavour must be utilised, for we know not from which quarter help may be forthcoming, whether from the biologist, the chemist, the physicist, the pathologist, or the clinician. The proper equipment of a cancer institute includes all these, and with such organisation as will get the maximum efficiency for each. I should like to give one word of advice and encouragement to the clinician. Yours is the brunt of the dreary day-by-day conflict with the disease. Do not merely watch eagerly for those isolated pieces that emerge periodically from the research laboratory, endeavouring to fit them into the therapeutic puzzle. And still less do not wait for a full revelation of the complete picture. It may be with you, after all, with your bedside observations, and with your efforts at treatment according to present knowledge, that the key of the problem of human cancer rests. Out of such observations and out of the results of such treatment may arise, not only treatment more perfected, but actual elucidation of the cause of the disease.

June 9th, 1913.

A Simple Method of Treating Epistaxis.—Pech.—Epistaxis being due to the rupture of the capillary vessels of the nose owing to congestion of the mucous membrane, the treatment is to lower the arterial tension while favouring coagulation of the blood at the bleeding point by exposure to the air. Lowering of the cerebral blood-pressure by prolonged forced inspiratory efforts in the following manner fulfils both these indications. The patient obstructs the nostril which does not bleed by pressing the ala against the septum with the finger, sufficiently firmly to reduce the opposite nostril by which the blood escapes to a slit. A forced inspiration, lasting five to eight seconds, is then made through this nostril, the mouth being closed, and the chest, when distended, emptied by a short expiration through the mouth. This process is repeated two or three times, preferably in the standing position, with the head erect. The fall in the cranial blood-pressure is so marked that to persist would produce syncope. Care must be taken not to detach the clot in the nostril and thus cause recurrence of the bleeding.—*Gaz. des Hopitaux*, p. 60.

THE CAUSES AND TREATMENT OF HEADACHE.*

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GENTLEMEN,—I show you to day a typical example of a disease possessing many features of interest, and one oftener encountered in private than in hospital practice, viz. migraine. The patient is a housekeeper, æt. 38 years. There is no family history of insanity, epilepsy, hysteria, migraine, or any other form of nervous disorder. She has suffered from measles, scarlatina, and diphtheria—all occurring in her twenty-fourth or twenty-fifth year. She has been neglectful of her diet, taking little solid food and living largely upon tea. She has been left much alone, and has had very little exercise or fresh air.

History of present illness.—From the age of fourteen she has been subject to "bilious attacks," characterised by visual disturbance, headache, and vomiting. These attacks occurred regularly every month, and coincided with her menstrual periods. From the first premonitions to the complete subsidence of the symptoms the attacks lasted about forty-eight hours, the acute symptoms about twenty-four hours, and her health in the intervals between the attacks was normal. For the past three months the attacks have become more frequent, and she now anticipates one every Wednesday.

Description of the attacks: (A) *Premonitory symptoms.*—The evening before the attack she feels chilly, no matter how warm the room, and she is listless, dull, and "inclined to be crying." Then "black specks and motes" appear before her eyes, changing to "larger black specks and black beads" and "dirty white pearls." Presently, there come "showers of these rolling down like a curtain and rolling up again," then "red and green stars," "yellow zigzags with red streaks at the top," and "yellow whorls and wavy lines."

A slight buzzing in the ears accompanies these visual phenomena. She has "a sort of broken sleep" until 4 a.m., when she wakes up with headache.

* A Clinical Lecture delivered at the Royal Victoria Hospital, Belfast.

(B) *The headache.*—This is very intense. It begins in front and "behind her eyes," and then "shoots into the back of her head." It "hurts her to look at anything." The pain is "all over her head." It is not confined to one side. It is succeeded by vomiting.

(C) *The vomiting.*—This is urgent, incessant, and accompanied by much nausea. Sometimes she vomits incessantly from 6 a.m. to 11 p.m. She says the vomiting occurs sometimes as often as every five minutes, so that "she could fill a bucket with it in the day." The vomited matter is usually yellow or pale green, frothy, and rarely contains anything but fluid. The vomiting subsides about bed-time.

(D) *Further progress of the attack.*—About midnight she falls into a broken sleep, waking now and then with some headache. Next morning the vomiting has quite ceased, leaving her very weak. In the afternoon the headache disappears, and she feels "inclined to sleep all the time." That night she sleeps soundly, and the following morning she is quite well.

This is a typical attack of migraine, and quite unlike any other affection.

A general examination of the patient elicits the following points: She is ill-nourished, weight 6 st. 2 lb. 12 oz., tongue is coated, appetite and digestion bad, much constipation.

The pulse-tension is 115 mm. The heart is normal. The knee-jerks are exaggerated on both sides. On admission ankle-clonus was present for a short time on the left side, but soon disappeared. Babinski's sign is not present. The right pupil is slightly dilated and a little sluggish in its reaction to light. The right optic disc shows some pallor. The left side is normal.

There is no record in these attacks of any form of paralysis or speech defect, or marked vertigo, or polyuria.

Since admission to hospital the attacks have recurred at the usual interval, but have been to a considerable extent abortive and controllable by remedies.

The pathology of this remarkable affection is unknown. We must assume an abnormal excitation of some cerebral area—the optic thalamus and the cilio-spinal region have been suggested—but neither the seat of the disease nor its precise nature has been determined. Whether the symptoms depend upon a "discharge" of nerve energy or some interference with the cerebral fluids we cannot say. The disease has some analogies with, and also some points of marked differentiation from, epilepsy. The various toxic theories of the causation of migraine are conjectural, and the vascular explanations are equally hypothetical.

The causes of headache are extremely numerous, and not easily classified upon any strictly scientific system. It is difficult, perhaps impossible, to avoid some degree of cross-division. The following classification aims at clinical convenience and practical utility:

Causes of Headache.

1. Toxic causes:
   - (*a*) Dyspepsia, including the various forms of gastro-hepato-intestinal disturbance.
   - (*b*) Renal disease: oxaluria.
   - (*c*) Rheumatism, gout, diabetes.
   - (*d*) Alcohol.
   - (*e*) Drugs, *e.g.* opium, morphia, salicylate of soda.
2. Reflex causes: irritation arising from—
   - (*a*) Teeth, gums, jaws.
   - (*b*) Eyes — hypermetropia, astigmatism, myopia.
   - (*c*) Ears.
   - (*d*) Nose.
   - (*e*) Ovaries and uterus.
3. Causes depending on nervous disease, functional or organic:
   - (*a*) Functional — overstrain, neurasthenia, hysteria, neuralgia, migraine.
   - (*b*) Organic—tumour of the brain, gumma, abscess, cerebral hæmorrhage, meningitis, neuritis of the nerves of the scalp, tabes dorsalis, syphilis of the central nervous system.
4. Circulatory and hæmic causes:
   - (*a*) Hyperpiesis.
   - (*b*) Anæmia, chlorosis.
5. Headache depending upon general infections:
   - (*a*) Enteric fever.
   - (*b*) Pneumonia.
6. Headache depending upon disease of the bones or soft tissues of the skull:

(*a*) Periostitis, osteitis, trauma.

(*b*) Erysipelas of the face or scalp.

This list, long as it is, might easily be further extended, but let it suffice for our purposes. It is a list suggesting obvious diagnostic indications, which we can only briefly discuss. Let us raise the various diagnostic problems by considering a series of hypothetical cases. As age is a factor of considerable diagnostic importance in connection with this subject let us take the cases in the order of time.

(1) *A child of from five to ten years of age suffers from persistent headache.*—Inquire for errors in diet, dental caries, intestinal irritants, constipation, hypermetropia, meningitis, enteric fever. These various causes can usually be disentangled without much difficulty. The food and the state of the bowels should be inquired into, the eyes examined, and the temperature taken. The headache of hypermetropia is frontal, usually supra-orbital, in seat; it is brought on or aggravated by school work; the letters may tend to run one into the other as the child reads, and there may be strabismus. The headache of meningitis is general in seat, it is persistent, often very intense, interferes with sleep, is often accompanied by vomiting and marked photophobia. This form of headache is apt to be ill-marked in quite young children. The headache of commencing enteric is frontal in seat, often severe, accompanied by a rising temperature and other evidences of the disease, and subsides somewhat suddenly, usually between the seventh and the tenth day of the attack.

(2) *An adolescent or young adult* (15–30), *male or female, suffers from persistent headache.*—Inquire for dyspeptic causes, dental caries, visual defects, anæmia, menstrual irregularities, cerebral tumour, meningitis, hysteria, rheumatism, enteric fever, renal disease. Most of these conditions are easily differentiated. Attention to the state of the digestive functions, the teeth, the eyes, the urine, and the temperature will clear up most cases.

Meningitis is difficult of early detection. There are no pathognomonic signs in the first stage. It is the assemblage and progression of symptoms which must guide us—the subacute onset, the fluctuating but significant pyrexia, the general hyperæsthesia, the persistent headache, the occasional emesis, the constipated bowels, the tendency to retraction of the abdomen, the state of the fundus oculi, and of the pulse.

Pneumonia is sometimes ushered in by severe headache, which has often led to the erroneous diagnosis of cerebral disease. A glance at the alæ nasi, an inquiry for sputum, a careful examination of the chest and due regard to the temperature should make this error nearly impossible.

Cerebral tumour may also for a time elude the most careful inquiry, but the persistent headache, the occasional vomiting, the changes in the fundus oculi, the general apathy, motor twitchings, and the state of the pulse will establish the diagnosis. Enteric fever should be inquired for if the headache is only of a few days' duration and accompanied by a rising temperature. The hysterical headache is often vertical in seat, extremely severe, or, at least, loudly complained of, and accompanied by other manifestations of hysteria. The rheumatic headache is often occipital in seat, and accompanied by tenderness of the muscles of the scalp.

(3) *A person in middle life* (35–55) *complains of persistent headache.*—Inquire for errors in diet, constipation, alcohol, renal disease, hyperpiesis, gout, rheumatism, gumma, tabes dorsalis, oxaluria, neurasthenia.

The habits of the patient, the state of the heart and circulation, the condition of the urine, and the nervous system call for special attention. Most of the conditions enumerated will be readily detected by systematic physical examination. The two conditions most likely to escape recognition are hyperpiesis and neurasthenia. The headache of increased arterial tension will probably be accompanied by some degree of arterio-sclerosis, and by some general evidence of cerebral congestion. Many of these cases give a history of free living and indulgence in alcohol. The headache of neurasthemia is vertical or occipital in seat; it is a dull ache or a sense of weight and oppression rather than acute pain; it is aggravated by fatigue, mental work, loss of sleep, and auto-suggestion.

(4) *A person in the decline of life* (60–80) *complains of persistent headache.*—Inquire for errors in diet, alcohol, gout, renal disease, oxaluria, hyperpiesis, drugs. The probability of circulatory causes for headache increases at this period. Many headaches in the aged are due to arterio-sclerosis. Gout, rheumatism and renal disease are important factors.

In the diagnosis of the causes of headache we naturally proceed from the more common to the rarer causes. Dietetic causes will have our first thought. The eyes, the urine, and the circulation must be interrogated. The possibility of headache being only a symptom of some general infection—enteric fever, pneumonia, tuberculosis—must never be forgotten. The state of the nervous system—other causes having been excluded—will demand attention and complete our survey. Most of the extraneous causes of headache are likely to escape detection only if their possibility is forgotten.

Most of the causes of headache are sure to occur to you, but I might condense my suggestions to you as follows:

In the case of children, test vision; examine the teeth; inquire for otorrhœa and put your finger on the mastoids; remember that the onset of tubercular meningitis is often very insidious; do not be misled by an abnormal onset of pneumonia; examine the urine.

In the case of adults, examine the urine; inquire for syphilis; test reflexes; examine the fundus oculi; do not overlook obscure cases of enteric.

In the case of the elderly, examine the urine; take the arterial tension; inquire into habits.

At all ages inquire into diet, the condition of the digestion, the state of the bowels; take the temperature.

If these rules are borne in mind, the explanation of any case of headache should seldom, if ever, escape us.

The treatment of headache is a large and important subject, which will hereafter often demand your attention. The table of causes will in many cases suggest a rational and obvious therapy. An aching or tender tooth will call for the dentist's services. Hypermetropia may be handed over to the ophthalmic surgeon. Syphilis of the central nervous system will require the usual specific remedies. Alcohol and drugs can be restricted or forbidden. Renal disease, oxaluria, rheumatism, pneumonia and enteric fever require general treatment, the relief of headache being a minor consideration. Meningitis and cerebral tumour will probably prove intractable.

Let us consider in some detail the cases where headache is the main feature, and its removal or relief a legitimate object of treatment. And to revert to our patient, let us begin by dealing with the treatment of migraine.

Migraine is notoriously intractable, though some relief can usually be obtained, and time is, on the whole, in the patient's favour, most cases showing some abatement with the progress of the years. That the disease is due to some kind of nervous instability may be safely assumed, and our first thought should not be of specific remedies, but rather of such general tonic and hygienic measures as are known to tend to fortify the nervous system. Rest, good food, fresh air, suitable exercise, plenty of sleep, freedom from worry, over-work or too much indoor confinement, hydrotherapy, travel—such are some of the measures most likely to be of service. Two cases have occurred in my private practice which illustrate the value of such measures. One is that of a clergyman of middle age and robust health, but formerly a martyr to migraine. He attributes his recovery to the practice of vigorous outdoor exercise, and especially to digging in his garden. The other case is that of a lady, highly strung, gifted and neurotic, whose health has been almost ruined by migraine. At home she suffers from incessant and regularly recurring attacks. Circumstances have enabled her for many years to obtain a two-months' holiday every summer, this holiday being usually spent in continental travel or at sea. During these holidays she enjoys complete immunity from her martyrdom. The inference from such cases is clear. We must brace up the general nervous "tone" if we hope to obtain even a partial victory over so inveterate a malady as migraine. In conjunction with such general measures as those just indicated, we should use general tonics or nervine remedies, according to circumstances, viz. iron, cod-liver oil, maltine, the salts of zinc, arsenic, bromides. Quinine is, I think, useless, and strychnine hurtful. For the relief of the paroxysm the best remedies are aspirin, antipyrin, phenacetin and caffeine. A combination of these remedies is desirable, and the relief obtained is great. Pressure over the temporal artery sometimes relieves. Some observers find the electric current useful. I have no experience of setons, which have been recommended. Psychical treatment, if it could be properly devised and wisely applied, would probably yield some benefit.

The headache of neurasthenia is often a large factor in the patient's protean miseries. It is a dull, wearying ache, vertical or occipital in seat,

aggravated by fatigue, mental strain, and auto-suggestion. We should, I think, avoid, if we can, any symptomatic treatment of this form of headache, and, in particular, we should be chary of the coal-tar derivatives. Bromides are permissible in moderation, and sometimes useful, but the essential point is the general constitutional treatment, according to the indications of the individual case. We should treat the neurasthenia (and that will often tax our powers to the full), and not the individual symptoms. Neurasthenics are bad subjects for any form of active medication. They stand drugs badly, just as they are intolerant of alcohol, tea, and tobacco, and they are easily injured by a too zealous hydrotherapy. Rest and mental distraction are the keynotes to the treatment of these troublesome cases.

The headache of hyperpiesis sometimes demands therapeutic measures. Any routine attempt to lower abnormally high arterial tension is to be deprecated, as we cannot feel sure how far such tension is one of Nature's protective devices, but there are cases where we may intervene with effect and advantage to our patient. A case for such interference is the following: A patient, usually of the male sex, age from fifty to sixty-five, who has eaten, drunk, and smoked too much, worked hard and lived hard, begins to suffer from headache, throbbing in the cerebral vessels, vertigo, and sleeplessness. His urine contains a trace, or more than a trace, of albumen, his aortic second sound is too sharp, and his pulse tension is 200 mm. or more. Such cases will bear depletion, and will improve under a light diet, abstinence from alcohol and tobacco, and diaphoretics and aperients. It is important not to overdo these depletory measures.

Ordinary neuralgic headache, oftenest involving the trigeminus, is common, especially in women, and frequently calls for treatment. The state of the teeth should be carefully investigated. I remember an instructive case which occurred in my practice a few years ago. A young lady suffered from trigeminal neuralgia, which proved most intractable. Her teeth were beautiful and apparently perfect, without any outward mark of caries. It seemed impossible to accuse them. But, after treatment had failed to give relief, it seemed to me that one molar, perfect to the view, was slightly tender on being sharply tapped with a tongue depressor. I recommended its removal—a course which the patient reluctantly assented to. It was removed, and there was no more neuralgia. The most successful medicinal combination in trigeminal neuralgia with which I am acquainted is one of croton chloral hydrate, gelsemium and bromide of soda. I have seen very striking results from these remedies given in conjunction. General tonics are useful, especially iron, and alcohol, which should be recommended with much circumspection, has a certain degree of utility. Local applications are not very successful. The ointment of aconitia is worth trying. Warmth to the affected parts is useful.

Rheumatic headache is not very rare, and seems to be usually occipital in seat. Salicylate of soda will be found useful, and the combination with it of bromides, as recommended by Dr. Little, of Dublin, assists its action.

In all forms of headache, rest, physical and psychical, is of the first importance. A good negative rule is to avoid opium, or its salts, if possible. However, in that terrible affection, tic douloureux, it may prove, as Trousseau insisted, our only resource.

Amongst remedies which have been recommended for headache, but of which I have little or no practical experience, I might mention the following: Acetanilide, belladonna, ergot, camphor, eucalyptus, kola, colchicum. Cannabis indica is, I think, a somewhat overrated remedy. This somewhat incongruous list of remedies is a reminder that the causes of headache are both numerous and protean, and that routine treatment is likely to be ineffectual. The key to success is the search for cause, and the careful differentiation of cases.

June 9th, 1913.

Treatment of Undescended Testicle.—Gorse and Swynghedauw have applied in nine cases with excellent results the following method: They incise the scrotum and introduce a large glass marble in children, a china egg in adults, and suture the skin over it. This is worn for two months. The scrotum has thus been prepared so that the testicle when brought down will find a practically normal bed and will have no tendency to slide upward. They do not have to stay in bed, but of course have to keep quieter than usual while wearing the smooth foreign body.—*Journ. A.M.A.*, vol. lx, p. 499.

A CASE OF SWELLING AND ULCERATION OF THE TONGUE.*

By LAWRIE McGAVIN, F.R.C.S.Eng.,

Surgeon to the Dreadnought Hospital, and to the Hospital for Women; Lecturer on Clinical Surgery in the London School of Clinical Medicine.

GENTLEMEN,—The patient before you is a man æt. 52 years; it is said that he is neither a heavy smoker nor inclined to alcoholism, nor has he, so far as he knows, had syphilis. Three weeks ago he noticed that his tongue was swelling and that he had developed a small ulcer on the left side of it. This has since become extremely tender and is rapidly enlarging, and he has difficulty in swallowing, and salivation is marked. He states that he had a somewhat similar attack some months ago. He is married, and his wife has had three normal confinements and two stillborn children.

He has had no illnesses of any importance. The case is one in which I would ask you to consider more especially the differential diagnosis, but before doing so let us go into its clinical aspect in detail. As the patient stands before you you will notice the drawn look on his face, the curious full appearance of his submaxillary and submental regions while his mouth is closed, and from time to time you will note the occurrence of an abortive effort at deglutition, which, from the facial twinge which accompanies it, is obviously painful. These signs are the outcome of a twofold effort: firstly, to make room for an enlarged and painful tongue, and secondly, to dispose of an excess of saliva, which but for your presence he would probably void. On opening his mouth the marked fœtor of his breath is apparent although his teeth are not particularly foul. Now, let me impress on you the very important necessity of *using your eyes first and your hands afterwards*. You will miss many of the salient points of a clinical case if you permit your fingers to take precedence of your eyesight. As the man's tongue lies in his mouth you will notice that the left half of it is dome-shaped in the posterior third, the dorsum touching the palate, while the right half is nearly its normal shape and size. Further, the affected half of the organ is glazed, and red behind, and somewhat furred in the anterior two thirds; it can be protruded fairly well beyond the teeth, and when in this position you will notice a superficial ulcer on the anterior surface of the left side of the soft palate. Its character is noteworthy. It is roughly oval and irregular in outline; its floor is pale yellow in colour and its margins clean cut; it is surrounded by a vividly hyperæmic areola. If now you draw the tongue gently over to the right side of the mouth, the lateral ulcer referred to by the patient is disclosed. This you will see occupies the middle third of the tongue. It is about the size of a shilling, oval, punched out, with a floor closely resembling that on the soft palate. In the resting position of the tongue it does not correspond in site to a ragged tooth, as you will notice that the upper jaw is edentulous, and that the first bicuspid tooth of the lower jaw on that side is well in front of the ulcer, and that there are no teeth behind it remaining in this side of the jaw. There is no hæmorrhage from these ulcers, nor any clot in the floor pointing to its recent occurrence.

Now turn your attention to the consistence of the tongue, and you will find, on drawing it forward and taking the swollen portion between the finger and thumb, it yields the sensation of elasticity but not of fluctuation, being firm but compressible, not hard and cartilaginous. The raised, but not everted, margins of the ulcers are firm but not hard, and tenderness is very marked, especially at the anterior end of the lateral ulcer. There is no induration of the surrounding tissues or in the floor of the mouth.

An examination of the neck reveals the presence of one or two large and fairly soft glands in the submaxillary region, but none in the deep cervical chain. They are movable, and are not fixed to the skin nor to the deep parts, nor is the skin over them discoloured. His temperature and pulse are normal. Between you, gentlemen, the following conditions have been suggested as alternative diagnoses:

(1) Acute septic glossitis and lingual abscess in association with a dental ulcer.

(2) Rapid epithelioma and acute glossitis.

(3) Tertiary gummata.

(4) Tuberculosis of the tongue.

* A Clinical Demonstration in the London Post-graduate School of Clinical Medicine.

(5) Actinomycosis.

Let us consider these in turn.

The suggestion of acute parenchymatous glossitis and abscess following dental ulcer has this in its favour—that the condition in this patient is of short history, and is accompanied by pain, tenderness, swelling and salivation. On the other hand, abscess formation in the tongue is more rapid even than in this case; it is commonly associated with marked and even dangerous œdema of the palate, fauces or epiglottis, which is here certainly absent; it is associated with marked pyrexia and constitutional disturbance; it is not common as a complication of dental ulcer; it does not usually give rise to glandular enlargement; it is more likely to be associated with the multiple small lesions of ulcerative stomatitis than with large excavating ulcers such as you see in this case.

Again, the suggestion that the lateral ulcer is the result of the irritation of a ragged tooth is not only doubtful, seeing that there is no tooth corresponding with it in position when the tongue is at rest in the mouth, but it leaves the presence of the second ulcer on the palate unaccounted for. This clearly has no connection with a dental ulcer.

The second diagnosis, that of rapid epithelioma, is not by any means impossible, for although the usual duration of a cancer of this size is about six months, I have twice seen the condition bear a close similarity to the case before you as far as the lateral ulcer is concerned. The points on which I would lay stress, however, as against such a diagnosis, are these:

(1) Epithelioma rarely, if ever, gives rise to secondary isolated masses in the tongue.

(2) It rarely, if ever, produces an abscess in this organ.

(3) It is always single, and in this case there are two well marked ulcers.

(4) The soft palate is not a common situation for epithelioma.

(5) When epithelioma is very rapid, glands are not as a rule large enough to be felt in the early stage. If, however, the ulcer is deeply excavating and the mouth is very foul, septic absorption and glandular enlargement may at times occur as early as this.

(6) Although not a very important point, the absence of any well-recognised pre-cancerous conditions of the tongue in this case, such as cracks, fissures, syphilitic leucoplakia, and chronic superficial glossitis, tends to negative the probability of cancer.

(7) The absence of pain shooting into the ear or side of the temple is rather against epithelioma.

(8) The fact that there is no family history of cancer in this case ought, I think, to weigh against such a diagnosis to some extent. I know that certain eminent surgeons are inclined to minimise the influence of heredity here, but I am not of this opinion. I have at the present time under my care a patient whose mother, sister, and one brother have all died of the disease in different situations; a second brother has had his tongue removed for the disease; while I have recently performed the same operation for the patient in question for recurrent papilloma. If this, gentlemen, does not bear on heredity, what is the reasonable explanation of it?

(9) Lastly, the comparatively free mobility of the organ is against the diagnosis of malignancy; but undue importance should not be accorded to this sign in this particular case, since the rapidity of the ulceration, presuming it for the moment to be malignant, is too rapid to give rise to sufficient extension to the floor of the mouth to bind the tongue down.

One of you has suggested the possibility of actinomycosis, and where you are prepared to consider "tertiary" syphilis you may reasonably discuss actinomycosis. There are, however, five points on which you may here exclude it definitely. Firstly, actinomycosis is exceptionally rare in the tongue of human beings (about 6 per cent. of all cases originating in the mouth), although much commoner in animals, when it is known as "woody tongue." Secondly, it starts in the anterior half of the tongue. Thirdly, it is a very slow process, extending over months as a hard mass or several nodules in the lingual substance. Fourthly, it breaks down late, producing multiple sinuses, *not ulcers*, discharging the yellowish-brown granules characteristic of the disease. And fifthly, it does not, as a rule, result in glandular enlargement.

Now consider for a moment the manner in which tubercle affects the tongue. In this situation it is commonly multiple, and originates, like tuberculous laryngitis, as a secondary manifestation of advanced phthisis. It is more especially confined to the anterior third of the dorsum, or the tip and under-

surface of the tip. There may be one or more small superficial ulcers, not as a rule central, and exhibiting the typical characters of the tuberculous ulcer, *i. e.* they have undermined, "mouse-nibbled" edges, a pale floor, devoid of granulations, or in which flabby, unhealthy-looking granulations are seen, and the discharge is simply a watery one. They do not bleed, they give rise to no induration, nor marked hyperæmic areola, and they are often exquisitely tender, so much so that feeding must be preceded by an application of cocaine or be carried out by the tube. The victims of such a condition, gentlemen, present a very different appearance than does the patient before you. Sallow, hectic, prone to perspiration, often with an excessive growth of silky hair along the spine and on the outer aspect of the arms and forearms, and with a lack of crispness and a tendency to loss of natural curl in the pubic, axillary, and anal hair; they frequently are the victims of anal fistulæ, the mouths of which closely resemble the ulcers in the tongue.

Only very occasionally is tubercle met with as a single ulcer on the lateral aspect of the tongue. I have not seen one for more than twelve years, but its character can seldom be for long in doubt.

You will see, then, that none of the above conditions satisfy the requirements of this particular case, and by the process of exclusion we are left to consider tertiary syphilis.

In the first place disabuse your minds of the idea that tertiary syphilis is improbable in the absence of any previous history or of outward evidences of the earlier stages of the disease. The more experienced of you must have seen many cases of undoubted syphilis in the tertiary stage in patients who have never exhibited either a primary or secondary manifestation.

The swelling in this man's tongue is rapid in history; too rapid for most epitheliomata, and yet not sufficiently rapid to suggest an acute abscess; it would fit in with the suggestion of gumma, but gumma almost invariably occupies the posterior third of the tongue and generally affects the middle line or lies immediately to one or other side of it: and the explanation of this is that syphilis, being a disease of connective and especially of fibrous tissue, and the median *raphé* of the tongue being the only fibrous structure in it, well marked only in the posterior third, it is in this fibrous *raphé* that the gummatous deposit is laid down.

Again, one of the chief characteristics of syphilis is the tendency of its lesions to exhibit multiplicity and multiformity: thus you will see at times a tongue affected at the same time by leucoma, superficial ulceration, and deep gummatous deposits. In the case before us you see two separate ulcers in association wirh a deep-seated swelling, a condition markedly at variance with the usual clinical signs of malignant disease. The character of the glandular enlargement is entirely in keeping with a diagnosis of tertiary syphilis, since these glands are large, soft, movable, and devoid of tenderness. The diagnosis then, gentlemen, is that of tertiary syphilis. It is obviously important to obtain absolute confirmation of this diagnosis, and that at the earliest possible moment, and for this purpose four methods are at your disposal. They are:

(1) The microscopical demonstration of the *Spirochæta pallida*.

(2) The Wassermann reaction.

(3) The reaction of the ulcers to large doses of potassium iodide.

(4) The microscopical examination of the margin of the ulcers.

Of these methods the first two involve little loss of time and give positive evidence of syphilis. As regards the third it is well to remind you that potassium iodide is at times capable of causing marked diminution of even malignant ulcers, and its action is therefore misleading; the effect is, however, always temporary and slow in the case of epithelioma, but rapid and permanent in that of gumma. The fourth method is rapid (in the case of a frozen section), and gives definite information, but I would caution you that at all times where you would employ it, you should first of all have obtained the patient's consent to immediate operation, since the effect of snipping out of portions of malignant growths frequently supplies a fierce stimulus to their activity, and converts a smouldering fire into a rapid conflagration. The best example I can give you of this peculiarity is to be found in the larynx, where such action may cause an intra-laryngeal epithelioma to sprout rapidly like a cauliflower and become extra- as well as intra-laryngeal.

The next point is important; the mass in the posterior third of the tongue may easily be an abscess, since many gummata break down early.

This may, of course, be ascertained by the use of an exploring needle, and presuming that pus is found, the question you will naturally ask yourselves is: "Am I wise in following the usual rule and incising when pus is present?" There are very few situations in the body, gentlemen, where you will *not* do so, but I make bold to say that this is one where you should at least hold your hand and do nothing rashly; and these are my reasons:

(1) The broken-down *débris* of a gumma is capable of being rapidly absorbed under the influence of large doses of iodides.

(2) The natural tendency of a gumma when incised to form in so soft a tissue as the tongue muscle a large mass of stinking granulation-tissue, primarily specific and secondarily septic, must be remembered. Once incised it is difficult to keep clean, and opens up a focus for sepsis which may result in œdema, septic pneumonia, etc.

(3) Provided no sepsis be introduced, a deep gumma of the tongue does not as a rule of itself tend to cause œdema of the glottis or fauces.

(4) Gummata in all parts of the body react much more readily to anti-syphilitics when in an unbroken state.

The indications, therefore, I would suggest to you for incision are these: firstly, commencing œdema; secondly, increase in size of the mass in spite of large doses of iodides; thirdly, the occurrence of constitutional disturbance, tenderness and salivation. It stands to reason, therefore, that there are cases which should be kept under close observation. They may not be dealt with as out-patients, since incision or even tracheotomy may be called for within a comparatively brief period.

The question of treatment by the injection of salvarsan is one on which it is, I think, as yet too early to express any very definite opinion. There is to my mind no question about the rapid resolution of many of these tertiary cases under its influence, especially when applied by the intra-venous method; but I am at present inclined to reserve it for cases of extreme urgency, and for those in which the usual anti-syphilitic treatment fails to control or lags unduly in its action.

June 9th, 1913.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from page* 144.)

(220)

Mixed Morbid Conditions,
or
The Mongrel Character of Diseases of the Chest.

I suppose we are all disposed to regard disease of the chest, and, in fact, diseases in every part of the body, as separate, independent morbid entities with little or no relation to each other. We acquired the habit in our student days, because inevitably in our text-books and in systematic lectures diseases have to be described separately. The practice still clings to us in later years, and we continue to speak of, and to diagnose, bronchitis, pneumonia, pulmonary tuberculosis, pleural effusion, etc., as though they had no connection whatever with each other. As a matter of fact, however, morbid conditions of the chest are more often combined than met with singly, and as a result either of mixed infections, simultaneous or consecutive, or of acute disease supervening upon the top of chronic, or of one morbid condition predisposing to, or giving rise to another, the most varied and ofttimes perplexing conditions are met with in the chest. Unless we are prepared to recognise the existence of these mixed morbid conditions we are certain to be led into serious errors in diagnosis, in prognosis, and in treatment.

("Mixed Morbid Conditions in the Chest," by J. Walter Carr, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxviii, p. 5.)

(221)

When to suspect Empyema after Pneumonia.

When, after an attack of acute pneumonia, the patient's condition remains unsatisfactory, not necessarily with a high temperature or any urgent symptoms, but still not improving, and when at the same time dulness of a most absolute character persists at one base, disregard the auscultatory signs entirely, always explore for pus, and generally in these circumstances you will find it.

(*Ibid.*)

(222)

The Marine Store Dealer.

Last winter I went down to Bromley-by-Bow to see the child of a gentleman, who appeared to be what is called a marine store dealer. I imagine that description covers a multitude of trades, except those connected with the sea. He had a very curious collection of miscellaneous property, which had found its way into his back parlour.

("Acute Bronchial Affections in Young Children," by T. D. Lister, M.D., F.R.C.S., 'Clin. Journ.,' vol. xxxviii, p. 12.)

(223)

Syphilis, one of the Diseases of Occupation.

First and foremost, "lest we forget," never omit to inquire carefully not only as to the patient's present occupation, but as to his past modes of employment. Though now, for instance, a farmer in a remote country-side, he may have previously been in one of the Services, or lived abroad. But let no occupation or social grade, high or low, cause you to disregard any otherwise well-founded suspicion of the specific nature of the lesion in question, since directly or indirectly, even the absolutely innocent may become the victims of infection. Thanks to the acknowleged difficulties of common-sense parental education in these matters, and a foolish sentimentalism in regard to State legislation, which refuses to face alternatives, under certain circumstances, as I have said elsewhere, specific infection may almost be included in the list of occupation diseases.

("Remarks on the Clinical History and Treatment of the less obvious types of Gummata, with records of eight cases," by Henry Curtis, B.S., F.R.C.S., 'Clin. Journ.,' vol. xxxviii, p. 23.)

(224)

In Cases of Obscure Disease never forget to look for the Handwriting on the Shins.

Experience at home and abroad leads me to believe that sometimes cerebral syphilis is ignorantly, or perhaps charitably, diagnosed as "cerebral malaria." Therefore be on your guard in all such cases; do not forget the possibilities, scrutinise the patient's record, look about for the stigmata of disease other than malaria. This is a hint you will find of great use in obscure disease not only of the brain, but also of the liver, for during the examination of cerebral or abdominal conditions one is apt to lose sight of the shins, where "the writing on the wall" may be so plain as to require no Daniel "to interpret, or dissolve doubts."

(*Ibid.*)

(225)

Doubting Thomas,

or

Fibroids under Twenty.

Between twenty-five and thirty fibroids are not particularly common, between twenty and twenty-five they are rare, and under twenty so rare are they that it is necessary to show the tumour, after its removal, together with a copy of the birth certificate of the patient from whom it was removed, before one can hope to convince the members of a medical society that one has met with such a case.

("Fibroids of the Uterus and their Complications," by Comyns Berkeley, M.A., M.D.Cantab., F.R.C.P.Lond., 'Clin. Journ.,' vol. xxxviii, p. 70.)

(226)

Errors of Refraction as a cause of Headache.

And I might, in passing, remind you to always think of the possibility of errors of refraction being a cause of headache. I have known a man sent round the world to try to get rid of a headache, and who, after circumnavigating the whole globe, came back with his headache just as bad. He then went to get glasses to cure his errors of refraction, and his headache passed away in a day. So never forget the importance of this: never see anyone with a headache without examining the refraction of their eyes.

("A Doubtful Case of Cerebral Tumour," by W. Hale White, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxx, p. 1.)

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THE TREATMENT OF ARTERIO-SCLEROSIS AND HIGH TENSION.*

By F. de HAVILLAND HALL, M.D., F.R.C.P.,
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Mr. President and Gentlemen,—As a physician, keenly interested in the subject of life assurance, I have been much impressed by the figures which have been brought forward to show that the great improvement in the general mortality of the country is becoming confined to the earlier years of life, but that after 45 the mortality has not improved of late years—indeed, between 45–65 there is an actual increase in the death-rate, For females the figures are somewhat better, there being still a considerable decrease of death-rate between the ages of 45 and 55, and a very small decrease continues until 85 is reached, but the gain is much less than in the earlier decades.

From the Registrar-General's reports it has been shown that, both in men and women, diseases of the heart and blood-vessels were the registered causes of about one third of the total deaths in the age-period 55–65. It is, of course, probable that this high mortality from diseases of the heart and blood-vessels is in part the consequence of rheumatic fever occurring in earlier life. A large share of it, however, is caused by degenerative changes occurring between the years 45 to 65, these changes being of an arterio-sclerotic nature. Before we can expect to cope satisfactorily with these degenerative changes we must have a clear idea of the conditions which tend to induce the early onset of arterio-sclerosis. In this respect certain factors have to be considered. In the first place I think that it will be conceded on all sides that we live faster than we did even twenty years ago. More work is expected of a man in a given time, and even as regards amusements they seem to be of a more

* Read at the Medical Society of London.

exciting nature than formerly. The advent of the telephone and the motor car has done much to increase the strain and stress of life. Can we, then, be astonished that there is a greater tendency to early degenerative changes when we consider this aspect of life. The increase in the amount of insanity is another evidence of the ever-growing pressure of business competition. Another factor in the occurrence of premature degenerative changes is that of the survival of weaklings, who would have succumbed under the harder *régime* of fifty years ago. A great many, therefore, reach adult life who would formerly have died from various infectious diseases and illnesses incidental to early life.

Then comes the important question of food. There can be no doubt that much more meat is consumed than formerly. The consumption of tea and coffee has also enormously increased. With regard to alcohol, there seems to me to have been a great change in the particular form taken. When I was a young man malt liquors were the general drink; now wherever you go and at any time of the day it is whisky—whisky. Lastly, I would draw attention to the ever-increasing consumption of tobacco. It is difficult to produce definite evidence, but I have no sort of doubt that excess in tobacco is a potent factor in the production of emphysema, arterio-sclerosis, and other degenerative changes; and in a recent conversation with Professor Schott he expressed the opinion that tobacco had a deleterious effect upon the cardio-vascular system.

I have very little to say about the *rôle* played by syphilis in causing arterio-sclerosis. Though the malignant forms of syphilis, which were not infrequently seen in my student days, are now of rare occurrence, the more the question of syphilis is investigated the more far-reaching is found to be its influence. That aortitis of syphilitic origin is a very common cause of aneurysm is now universally accepted, and similar changes are met with in the smaller arteries.

Huchard* has given some very interesting statistics on the ætiology of arterio-sclerosis. Out of 15,000 personal observations he has been able to sift 2680, with the following results:

Gout (with gravel and lithiasis)	693
Rheumatism	355
Syphilis	336
Alimentary	315
Tobacco	188
Infectious diseases	77
Diabetes	76
Alcohol	52
Malaria	42
The menopause	24
Moral and nervous causes	21
Doubtful cause	501
	2680

Plumbism, the pathogenic action of which resembles that of gout, does not figure in these statistics, because lead-poisoning is rare among the patients frequenting Huchard's consultations.

From what has been said of the ætiology of the condition we are discussing it is obvious that the all-important thing is to recognise the onset of arterio-sclerosis, and to take prompt steps to endeavour to arrest its progress. *Principiis obsta* should be our motto.

In the first place, the question of mental strain must receive careful consideration. Of course if the high tension be accompanied by signs of cerebral congestion, then absolute rest from all mental work must be enjoined, and work should only be very cautiously and gradually resumed.

The difficult cases to advise are men with high tension, leading strenuous lives of great mental activity. I endeavour to meet the requirements of the case by suggesting that the patient should leave off earlier than he has been accustomed, and take an afternoon off every week, in addition to the whole of Saturday. It is, however, impossible to lay down any general rule; each case must be decided on its own merits.

In the next place comes the question of diet. Auto-intoxication plays a very important part in the production of degenerative changes in the tissues generally, but especially in the arteries. Most people over fifty eat too much, and particularly too much butchers' meat. While writing this I was consulted by a gentleman eighty-three years of age, who said that he had three meat meals a day. His pressure was 202 mm. of mercury—a very suitable subject for an apoplectic stroke! The first direction I give to a patient suffering from arterio-sclerosis and

* Paper read before the International Medical Congress at Buda-Pest, 1909.

high tension is that the amount of meat eaten must be reduced. In advanced cases I advise that meat and soups made from stock should be discontinued entirely. The diet should consist of fish, poultry, vegetables, cheese, milk, oatmeal, bread, and farinaceous puddings. Honey has been recommended. Some patients derive much benefit from soured milk. Half a pint to a pint may be taken daily.

I am convinced that excess of salt is injurious, so I, therefore, limit the amount of salt and forbid salted foods. But not only the quality of the food eaten should be considered but also the amount. The patient should endeavour to reduce gradually the quantity of food to as little as is sufficient to keep him in good condition. If the patient be instructed to eat very slowly and to thoroughly masticate his food, he will find that he is satisfied with a smaller quantity of food than would otherwise be required. Should the patient be obese, then there will arise certain difficulties about the diet. Sugar should be replaced by saccharin or saxin. Potatoes, turnips, carrots and parsnips should be eschewed. Crisp toast, plasmon, or gluten bread should be taken instead of ordinary bread. Among biscuits I have found kalari, akoll, apax and spartan useful.

The amount of tea and coffee should be strictly limited. Black coffee should, of course, be prohibited. If possible, I get my patients to abstain from alcohol, though in the case of those who have a feeble digestion, or who seem much upset by the deprivation of all alcohol, I allow a small quantity of whisky, say, an ounce and a half daily, or an equivalent quantity of a dry sherry or moselle.

I lay great stress upon the importance of drinking an abundant supply of water. I think it very desirable that this should be free from chalk. I therefore order salutaris, or tell patients to get a still, so as to be able to provide their own distilled water. I recommend that a pint and a half should be drunk daily, apart from meals, and preferably hot. If the flat taste of distilled water be objected to, it may be flavoured with a little lemon-juice.

Regarding tobacco as one of the factors in the production of arterio-sclerosis, I advise that less than two ounces should be smoked weekly, and if the patient can be induced to give it up entirely, so much the better. In cases of tachycardia, palpitation, angina, or where the left ventricle is dilated, tobacco should be absolutely excluded. It is very striking how speedily improvement may ensue if tobacco be entirely discontinued. While I was writing this paper, a medical friend consulted me on account of cardiac discomfort and irregularity of heart-action. His impulse was in the nipple line, fifth interspace. I advised him to give up tobacco and coffee, and when I saw him six weeks later all his cardiac symptoms had disappeared, and the impulse was nearly an inch inside the nipple line.

After diet comes the consideration of what may be termed general hygiene. The condition of the skin requires careful attention. The cold bath is inapplicable to the class of patients we are considering, but a daily tepid or warm bath will be of great advantage. The question of Turkish baths often crops up. If the patients have been accustomed to them, and they have not been attended with any ill-effects, then I allow their continuance, provided the patients do not go into the hottest room. For a patient with arterio-sclerosis who has not been used to these baths, the risk, in my opinion, is too great. I would not, therefore, countenance a patient of mine commencing Turkish baths if he suffered from arterio-sclerosis of at all an advanced nature. Electric-light baths will sometimes be found to act most beneficially, but, like the Turkish baths, they require to be used with great discretion.

In these days it seems hardly necessary to advocate the importance of fresh air, but some people getting on in years have a great dread of contracting a chill, so that it may be advisable to see that they do not shut themselves up too much. Residence at the sea-side is not to be advised, and altitudes above 1500 or 2000 ft. are not desirable. Whenever practicable, exercise should be taken in the open air. Walking is the best form of exercise, but those accustomed to ride may be advised to continue to do so as long as possible—"The outside of a horse is the best thing for the inside of a man." Golf may be permitted, unless it fatigues the patient too much, or he is suffering from an advanced form of the disease. Should the patient for any reason be unable to take sufficient exercise, general massage will be found to be a very efficient substitute. Inasmuch as most of our patients have "fall'n into the sear, the yellow leaf," and have arrived at the time of life when the prostate is a trouble, it behoves us to pay particular attention to the state of the bladder. The mental unrest and irritation,

together with loss of sleep at night, caused by hypertrophy of the prostate and consequent inability to empty the bladder, will greatly aggravate the original disease. This is more particularly the case if the urine becomes septic from retention. Prostatectomy should therefore be advocated as soon as the bladder difficulty demands it.

Speaking of sepsis reminds me of the importance of attention to the condition of the teeth and thorough treatment of any existing pyorrhœa.

The possibility of effecting much benefit by medicinal means is small; nevertheless there are certain indications for this line of treatment. In the first place I would lay stress on the extreme importance of a daily action of the bowels without straining, *i. e.* we should endeavour to cultivate a loose rather than costive habit. The ordinary mild aperients, *e. g.* cascara, compound liquorice powder, and the colocynth and hyoscyamus pill may be employed. If the patient has high tension great benefit will result from an occasional dose of calomel or blue pill. Some patients do best with a dose of one of the mineral waters, such as Hunyadi Janos, Carlsbad or Friedrichshall, or a teaspoonful of Epsom or Glauber's salt in a claret-glassful of hot water in the morning. Of late I have been ordering a dessert-spoonful to a table-spoonful of pure paraffin at bedtime, and repeating the dose, if necessary, in the morning. This seems to answer in many patients with the minimum amount of discomfort.

The drug which I have found most generally useful in cases of arterio-sclerosis associated with high tension, is iodide of potassium. Unfortunately many patients are intolerant of this drug, and, indeed, of any iodine preparation. If iodide of potassium cannot be taken, iodoglidine may be tried, and will sometimes be tolerated when the iodide is impossible. One of my patients, a medical man, speaks highly of the effect in his own case of iodipin tablets. Each tablet represents one grain of potassium iodide. In patients who stand the iodide well it may be continued for years. At present I have under my care a lady, now æt. 70 years, who had an attack of an apoplectic nature followed by slight coma lasting twenty-four hours, and accompanied by retinal hæmorrhages and high arterial tension upwards of six years ago. Ever since she has taken a small dose (3 gr.) of potassium iodide daily, and when her tension rises and she suffers from headache, the dose is given two or three times a day. Under this line of treatment, coupled with careful dieting, the patient has maintained a fair degree of health, she has had no return of the apoplectic symptoms, and there has been distinct clearing up of the retinal hæmorrhages.

The iodides are very useful in the early stage of the disease, especially if it be accompanied, as is usually the case, by high arterial pressure. In restless, excitable people, and particularly in those who suffer from insomnia, the addition of a small quantity of bromide to the iodide mixture will be found beneficial, or a nightly dose of bromide of potassium or ammonium may be ordered. The combination of iodide and bromide is of a great service in the high tension so common at the menopause.

In some patients, even at the commencement, the pressure is low, and to these patients the iodides are rather harmful than useful. In most patients there comes a time of failing compensation, when the iodides are badly borne. Then we shall have to consider whether or no cardiac tonics are indicated. If the heart's action be frequent, digitalis or strophanthus should be ordered. As a rule I prefer the latter drug in combination with strychnine, but if patients do not respond to it, digitalis must be given. I have found Nativelle's digitaline in granules, gr. $\frac{1}{240}$, night and morning, very useful. Where there is simply a feeble action of the heart, without any increase in frequency, I have found the combination of arsenious acid and the hydrochloride of strychnine, one thirtieth of a grain of each, in a pill, given three times daily after meals most serviceable. The glycero-phosphates are also useful as a tonic; the preparations of iron are not well borne. With the onset of dropsy, one of the theobromine compounds such as agurin or diuretin may be given with advantage.

In the obese, small doses, *i. e.* 2 gr. daily, of thyroid extract are most beneficial in reducing weight and relieving excessive tension. If the drug suits the patient the dose may be cautiously increased, but I have never given more than 5 gr. three times a day, and in cases of arterio-sclerosis it is seldom advisable to give more than half that dose.

In a thoughtful paper communicated to the 'Practitioner' last August, Mr. T. B. Scott, of

Bournemouth, draws attention to a remedy, which he regards "as a safe and sure depressor of tension." He refers to the hippurate salts. They are readily soluble, and can be given in 1- or 2-gr. tablets. The lithia salt is more powerful than the ammonia salt. Of the former 3 or 4 gr. may be given daily, of the latter 6 or 7 gr. A patient, æt. 65 years, who is under my care at present, illustrates the good effect of the drug. When he started taking 2 gr. of the hippurate of lithia his pressure was 186 mm.; he was suffering from attacks of shortness of breath and anginal pains. A fortnight later his pressure had fallen to 176 mm. I increased the dose to 2 gr. twice daily, and when I last saw him his pressure was 162 mm., and he was in all respects much improved.

Guipsine, obtained from the mistletoe, has recently been recommended in cases of arterio-sclerosis. It is said to lower arterial tension due to central vaso-motor action without any depressing action upon the heart itself. I have never used it.

There is one remedy used in the treatment of atheroma and arterio-sclerosis about which I should like some information. I allude to Trunecek's artificial or inorganic serum. It is improperly called a serum as it does not contain any albuminous compound. It also goes by the name of antisclerosin. Its composition is sodium sulphate 44, sodium chloride 492, sodium phosphate 15, sodium carbonate 21, potassium sulphate 40, water *q.s.* to make 10,000. Trunecek advises that the solution should be given hypodermically, but it can be given by the rectum or mouth. It is said to be useful in the early stages of arterio-sclerosis, but useless in the later stages with dilated heart and asystole.

It is claimed that antisclerosin can remove or improve a number of the unpleasant symptoms of sclerosis, but even the warmest advocates for its employment do not pretend that it can cure sclerosis, which is as little curable as the emphysema which usually accompanies sclerotic changes in the blood-vessels.

Tablets of Trunecek's serum (anti sclerosin) are prepared (Martindale). Daily dose, three to six one hour before meals. Unpleasant effects are said to be of rare occurrence, but now and then vomiting has resulted. Antisclerosin may be given advantageously alternately with iodide of potassium, a fortnight at a time. For patients with the rheumatic diathesis the salicylate of sodium in combination with bicarbonate of sodium will be found of great service.

It will be noted that so far I have said nothing about the more powerful vaso-dilator group of drugs. I must preface my remarks about them by saying that I am of opinion that they have been used very indiscriminately and injudiciously in the past. It must be realised that the high pressure met with in these cases is an effort of Nature to maintain the balance of the circulation under adverse circumstances, and to order a vaso-dilator, simply because the sphygmomanometer registers, say, 200 mm. of pressure is the height of folly, and may be attended with disastrous results to the patient. On the other hand, there are certain indications for the use of these drugs which need recognition. The first and the most important is the occurrence of anginal symptoms. In cases of this kind the vaso-dilators may be given with great advantage. In very severe cases nitrite of amyl may be inhaled and its action may be kept up by the internal administration of nitro-glycerine. Then I have come across a series of patients who, on starting for a walk, especially in cold weather, feel a sense of constriction across the chest, sometimes accompanied by cardiac pain. These people are often made quite comfortable if they take, shortly before starting to walk, a tablet of erythrol-tetranitrate gr. $\frac{1}{4}$. I also tell these patients to carry about with them two doses of the following mixture: Liquoris trinitrini, ♏ $\frac{1}{2}$; tinct. chloroformi co., ♏x; sp. ætheris nitrosis, ♏xx; aq. ad. ℥ss. If at any time they feel the cardiac discomfort they can take a dose and repeat it in a quarter of an hour if necessary. I have found that a small dose of trinitrine acts better in solution than when given in a tablet. Another indication for the use of trinitrine is the occurrence of headache. As a rule nothing answers so well in these cases as a minim of the liquor trinitrini, preferably given in solution. In cases of persistent high tension accompanied by anginal symptoms or headache, not relieved by iodide of potassium, I have found nitrite of sodium in doses of 1 to 2 gr. three times a day, given alone or in combination with iodide of potassium, very useful. If the vaso-dilators are given symptomatically and not as a matter of routine, great good, and little or no harm,

will result from their administration. There is yet another remedy to which I must direct attention, and that is bleeding. I have seen the greatest possible benefit result from the abstraction of six to eight ounces of blood by venesection, or from the application of six to twelve leeches, bleeding being encouraged. Blood-letting is indicated in cases of high tension, with headache, confusion of thought, giddiness or other symptom suggestive of cerebral congestion. Spontaneous hæmorrhage from the nose or other organs should be looked upon as beneficial, and no measures should be taken to arrest it unless it be very profuse and the patient is manifestly becoming faint.

To people who can afford the time and money a course of spa treatment can be recommended with confidence. Among our own resorts I have found Harrogate, Llandrindod and Strathpeffer the most beneficial, though I have also seen very good effect from Woodhall Spa. On the Continent, Contrexeville, Vittel, Vichy and Kissingen are the places of which I have most experience. For the obese, Marienbad and Carlsbad are especially indicated.

Lastly, I must say a few words about the Nauheim system of treatment, both as practised at Nauheim and also as carried on in this country. I have seen great benefit resulting from its employment in some cases, and on the other hand, I have seen patients whose lives, I feel certain, have been shortened by a course of this treatment. It must not be regarded as a panacea for all kinds of cardiac degeneration, but the cases in which it is employed must be selected with great care, and the treatment should be under the supervision of a doctor who has made a special study of it. When discussing the effect of the Nauheim treatment with Dr. Schott, he pointed out to me that patients who had not responded to digitalis were often benefited by the drug after they had had a course of baths. He deprecated the employment of this method of treatment in cases of aneurysm, except in the very early stages of the disease.

Discussion.

Sir Lauder Brunton said that they all had listened with very great pleasure to the admirable summary of the treatment of arterio-sclerosis and high tension which Dr. Hall had given them. There were many points in the opener's paper which they might take up in discussion, and one of the most important related to the question of blood-letting, the reasons for it, and the *modus operandi*. The speaker at one time used to take it that blood-pressure was the product of two factors—first, the amount of blood sent into the arteries by each ventricular contraction, and next, the amount of blood passing out in the same time through the arterioles into the veins. But there was a third fact to be considered, namely, the condition of the blood-cells, or the viscosity of the blood. The importance of this factor of viscosity, so often overlooked, was brought before him in a striking case a few weeks ago. The case was that of an old lady with very high tension and renal inadequacy. One day he called to see her and found her in a state of coma. This he did not regard as being due to the brain; there was no evidence of local paralysis. He advised her doctor to open a vein and let blood. A bandage was thereupon put over the arm, but the vein would not swell. One could hardly see where to put the lancet. The muscles of the forearm were squeezed, and still the vein would not swell. After a great deal of trouble it became big enough to be seen, and on puncturing it the blood began to flow, but only in drops and very slowly. The blood was extremely dark. He noticed that the patient had got Cheyne-Stokes' respiration, and the difficulty in the flow of blood did not appear to be due either to the heart or the vessels. He knew from experiments that venosity of the blood increased its viscosity, and he thought that if he lessened the former he would lessen the latter. A cylinder of oxygen was standing near, and this he administered to the patient. Within a minute or two of inhalation the blood began to flow easily, and within five minutes it flowed readily in a spurt from the vein. He had had a similar experience with regard to the circulation in the lung. Some years ago he was watching a case of cardiac "asthma." The tension was down to 75 mm. Hg. in the radial artery. He gave the man oxygen, and within ten minutes the tension in the radial artery had gone up to 150 mm., showing clearly that blood was passing much more readily from the right side of the heart to the left. Many years ago, before the discovery of nitrites and iodides, the only remedy that was any good in cases of high tension was blood-letting, and he was very glad to hear Dr. Hall recommending it so strongly. It was really a

very good old remedy, now out of fashion, but it would come into fashion again, and that before long. It was worth while to consider the *modus operandi* of blood-letting. One would naturally think that by letting blood out of the veins one was diminishing the quantity in the vessels, and thus lowering the tension. But experiments had shown that fluid was absorbed very rapidly indeed from the tissues, so much so, that even after a large amount of blood had been let out the quantity of blood in the vessels soon became nearly equal to the normal. But although this was the case, the quality of the blood was not the same. The number of corpuscles in the blood was greatly diminished, and it took a considerable time before this number was restored. They were getting very wise in these days, but physicians long ago were very wise as well. He believed, indeed, that in the time of Hippocrates plethora was looked upon as the cause of apoplexy, just as they regarded it to-day. It was recognised long ago that one of the great things to do in keeping down blood-pressure was to pay attention to the condition of the blood. In the first edition of Watson's 'Physick' the three things necessary in the treatment of plethora were stated to be blood-letting, scanty feeding, and exercise. These were the very things that Dr. de Havilland Hall had recommended that evening. The speaker wished to impress upon them the necessity of attending very carefully to the exercise of the patient, and to keep him underfed rather than overfed. There was one other thing upon which he would lay stress, namely, the necessity not only of daily elimination from the bowel, but of the use of some mercurial aperient. There was one old remedy figuring in the Pharmacopæia of which mention should be made, and he recalled the fact that poor old Dr. Plummer had found out the value in these cases of high tension of a compound pill of calomel and antimony. Even now he thought this might well be of avail, allied to the blood-letting and low diet and exercise which Dr. Hall had recommended.

Dr. A. P. LUFF most cordially agreed with what Sir Lauder Brunton had said as to the excellent way in which the discussion had been opened. His own contribution to it would be very brief. He wished in the first place to lay stress upon the fact that they must not consider arterio-sclerosis and high tension as necessarily synonymous terms. He himself had seen amongst a number of gouty patients many cases of arterio-sclerosis, but high tension was very frequently absent. It seemed to him that gout specially conduced to that condition of atheroma which they so frequently called arterio-sclerosis, but in many of these gouty patients he had found quite a low tension, very frequently under 160 mm. Hg. Dr. Hall had laid stress upon one very important factor in the development of both arterio-sclerosis and high tension, namely, strain. This did not necessarily mean intense muscular efforts, but it might apply, as he wisely pointed out, to the increased strain and stress of modern life. Here he would remark that arterio-sclerosis and high tension had been put down hitherto as being common in men, while women, freed as they were from active participation in this strain and stress, suffered to a much smaller degree from trouble with the arteries. Nevertheless, many a militant suffragette must be laying up for herself a rare crop of arterio-sclerosis and high tension, and if women expected to secure men's rights they must be content at the same time to accept men's wrongs, and among them arterio-sclerosis and high tension. With regard to the toxic agents which were the cause of arterio-sclerosis and high tension, he only wished he could support Dr. Hall in ascribing these effects to tobacco and alcohol, but he confessed that from the results of his own experience he was unable—unfortunately unable—to agree with his contention that these were direct causative agents in the production of these conditions. The toxic agents he had met with most frequently were firstly those produced in the bowel by the excessive consumption of food, especially animal food, and with regard to that he would lay stress upon the importance of attending in the first place to the alimentary tract. The choice, the regulation, and the diminution in the amount of food were most potent factors in treatment. With regard to exercise, he quite agreed with Dr. de Havilland Hall that a suitable amount of exercise was most important, and walking was perhaps the best form for it to take. Dr. Hall, however, had mentioned one form of exercise in particular, namely, golf, and considering how very popular this was becoming, he did wish to utter a word of caution. He spoke as a golfing enthusiast himself, but he thought they ought to be careful as to indiscriminately advising golf for arterio-

sclerotics. Judging by the amount of temper over the game displayed by some of his acquaintances, and in view of the fact that high tension was frequently associated with anger, and if anger was to be measured by the strength and abundance of language which accompanied it, then golf was certainly not a game for the class of patient who could not control his temper. So far as drugs were concerned, he agreed with Dr. Hall as to the great importance of two, namely, iodide of potassium and the nitrite derivatives. But he agreed also that in cases of advanced arterio-sclerosis, unless associated with anginal symptoms, it was wrong to administer vaso-dilators. Although Dr. Hall had mentioned spa treatment, he had not referred to one cognate subject, namely, the effect of climate. If it was desirable to send these patients away, then it was important to avoid a bracing climate with a very low relative humidity. Egypt and the mountains were places to which these patients should never be sent. In England they were fortunate in possessing places, both on the coast and inland, having a high relative humidity, combined with other advantages suitable for this class of cases. Two were Torquay and Sidmouth, and similarly favoured climatically were two inland resorts, namely, Bath and Cheltenham.

Dr. W. Bezly Thorne said: In approaching the subject of arterio-sclerosis and high tension I have in mind two types of morbid process which not uncommonly are classed together, but which are in some respects essentially distinct. The one might be described as the atonic and atrophous, tending to dilatation of both heart and vessels; and the other as the hypertrophous, with a tendency to loss of lumen or obliteration and to compensatory cardiac hypertrophy. The latter in some degree tends to approximate to the former when the stage is reached in which degeneration invades the hypertrophied myocardium and undermines its conservative and beneficent influence, and there are intermediate cases.

The atonic type is marked by a low balance, or so-called diastolic grade of pressure and a high maximal grade, the one standing at from 80 to 100 mm. Hg., and the other at from 100 to 200 mm. Hg., the average difference being about 150 mm. In the hypertrophic and obliterative type the lower readings may be from 110 to 180 mm., and the higher from 200 to 250, and in exceptional cases as high as 300 mm., the average difference between the two readings being about 60 mm.

In children and young people the atonic type is generally marked by warm extremities, and a full, soft, and somewhat dicrotic pulse tending to undue frequency; while in the hypertrophic and obliterative type the extremities are cold, bluish-red in colour, subject to chilblains, and in extreme cases the patients display alternating syncope and asphyxia, as seen in the early stages of Raynaud's disease. The pulse is small and tends to be incompressible. I cannot help thinking that the late Dr. George Balfour had both these types in view when he wrote of the senile heart in childhood. The two types of affection may be readily distinguished at all ages by making sphygmometric records in the grades which I have indicated. I gave my reasons for so doing in some detail last year, in the course of the discussion of Dr. de Havilland Hall's paper on "The Sphygmometer in General Practice," but unfortunately I am not able to refer you to them, as by some accident my contribution was not included in the Society's 'Transactions.'

In the atonic type toxæmia is the fundamental condition. It may be malarious or pyogenous, but in the great majority of cases it is of alimentary origin, and mostly associated with some catarrhal interference with the patency of the common bile-duct or its orifice. The following are examples of the influence of alimentary toxæmia on blood-pressure in such cases:

Case No. 1.—*Man, æt. 50 years.*

	Pulse.	Balance or diastolic pressure.	Radial tactility ceases.	Brachial obliteration.
March 15th, 1913	84	100	150	170 mm. Hg.
April 16th, 1913	66	80	110	125 ,,
Fall . .	18	20	40	45 mm. Hg.

The first was the case of a man, æt. 50 years, with obvious symptoms, which I will not enter into, of alimentary toxæmia. The readings when he was first seen on March 15th are given in the table. One of the features of the treatment in this case, in addition to the dieting, was an emulsion of paraffin with bismuth. Within little more than a

CASE NO. 2.—*Man, æt. 57 years.*

	Pulse.	Balance or diastolic pressure.	Radial tactility ceases.	Brachial obliteration.
August 16th, 1912	82	90	150	220 mm. Hg.
,, 23rd, 1912	72	80	160	170 ,,
	Fall 10	Fall 10	Rise 10	Fall 50 mm. Hg.
April 14th, 1913	68	80	155	175 mm. Hg.
	Fall 14	Fall 10	Rise 5	Fall 45 mm. Hg.

Adrenalin and strychnine, June 28th, 1912.

month the readings showed the fall set down in the table. The second case was a man, æt. 57 years, who had a systolic murmur at the apex and in the aortic region. Within seven days, under somewhat similar treatment, his pulse had fallen by 10, his diastolic pressure had fallen by 10, his radial tactility showed a rise of 10 (into the reasons for which I will not enter), and the brachial obliteration had fallen by 50 mm. of mercury. I saw him again about eight months later, when, compared with the original reading, his pulse showed a fall of 14, his diastolic pressure a fall of 10, his radial tactility a rise of 5, and his brachial obliteration a fall of 45. And this man, who had been suffering from dyspnœa, tremor, præcordial and anginous pain, was able to lead a life of moderate activity without any other treatment.

The hypertrophic type is less common, and it is not always easy to trace its genesis, but it is certainly liable to be influenced in its progress by alimentary toxæmia. Syphilis, gout, abuse of alcohol and of tobacco, and the fibrositic tendency seem to be causatively associated with it. Of lead poisoning I cannot speak from personal experience. With regard to the fibrositic tendency, although it may be noted in both classes of cases, in the hypertrophic that process seems to have a special incidence on the circulatory mechanism.

As regards treatment, the measures to be employed are those which may be depended on to effect some degree of repair while at the same time lowering blood-pressure, and of these the one which is primary and fundamental is the correction and avoidance of toxæmia. Alimentary autotoxis is by far the most common primary cause. Where the patency of the common bile-duct is interfered with, the fundamental conditions on which the biochemistry of intestinal digestion depends are wanting, and morbific bacterial action throughout the intestinal tract is insured. I rely largely in these cases on limitation of carbohydrates, fractional doses of mercury in some form, and, above all, on an alkaline emulsion of paraffin with bismuth taken with a draught of warm water an hour before meals. The treatment for malarious, syphilitic, and pyogenous toxæmia need not be entered on, except to observe that in all doubtful cases recourse should be had to the Wassermann test. Neither need the value of the iodides be enforced; but I cannot refrain from drawing attention to that iodide of fibrolysin which we owe to Cognet, and which is commercially known as T. iodine. It can be given in 5-gr. pills two or three times a day, or better still, be intra-muscularly injected two or three times a week, and later once a week.

The following example is that of a patient whom I have seen from time to time with Dr. Gilbert Smith, of Hindhead. Man, æt. 67 years:

	Balance or diastolic pressure.	Radial tractility ceases.	Brachial obliteration.
July 13th, 1912	190	290	300 mm. Hg.
October 12th, 1912	170	240	260 mm. Hg.

(Thirty-six injections.)

Saline baths administered a few degrees below the temperature of the body and of a strength rising from 2 to 8 or 10 per cent. of salt are often efficacious in lowering pressure while relieving fibrositic and rheumatoid conditions.

Mr. Hewer, of Stratford-on-Avon, informs me that in the case of a patient, æt. 73 years, whom I have treated in conjunction with him, simple saline baths, in three weeks, induced a gradual fall from 170 mm. (Riva-Rocci) to 140.

The Nauheim baths and exercises offer in the highest degree the means of simultaneously lowering blood-pressure and effecting cardio-vascular repair. They would appear to increase the supply of reparative material through the vasa-vasorum as well as the coronary system, and even in advanced cases, in which the pressure cannot be lowered, compensatory repair may be set up which will enable the patient to return to a moderately active life, to which dyspnœa, insomnia, commencing œdema and anginous pain seemed to have finally

closed the door. I could quote many such cases, but the following is the more interesting as there was no great fall in blood-pressure from the initial record in 1903 up to 1909.

November 5th, 1903:

C—, æt. 63 years, retired colonel, Army Medical Department. Tremor cordis, palpitation, anginous pain on exertion, radials closely beaded. Before treatment, pressure 170–220 mm. Hg.; after treatment 140–200 mm. Hg. All symptoms except beading of radials relieved.

May 31st, 1904: Pressure 120–150 mm. Hg. July 18th, 1904: Pressure 120–140 mm. Hg. Feels quite well.

June 22nd, 1905: Pressure 120–140 mm. Hg. July 27th, 1905: Pressure 120–140 mm. Hg. On days of sultry heat the maximal readings were uniformly about 20 mm. higher.

June 16th, 1906: Pressure 140–170 mm. Hg. July 11th, 1906: Pressure 140–160 mm. Hg. Feels quite well.

June 7th, 1907: Pressure 130–150 mm. Hg. July 4th, 1907: Pressure 130–150 mm. Hg. On sultry days maximal readings 20 mm. higher. Feels quite well.

May 4th, 1908: Pressure 130–160 mm. Hg. June 4th, 1908: Pressure 140–175 mm. Hg. Feels quite well.

June 3rd, 1909: Pressure 150–220 mm. Hg. June 24th, 1909: Pressure 130–200 mm. Hg. Feels quite well.

No further bath treatment.

January 1st, 1913: The patient reports, "I have been wonderfully well. I think I feel better and stronger than I did ten years ago."

The beading of the arteries underwent no change. The rise of pressure in sultry heat is noteworthy. The treatment consisted of Nauheim baths and correction of alimentary toxæmia.

As to vaso-dilators of the nitrite order, it remains to be said that their routine and persistent administration is to be deprecated. Their effect is very transient, and they induce no repair. On the contrary, their persistent administration weakens the myocardium and induces paresis of the tunica media, and if they do succeed in lowering blood-pressure they hasten the onset of, or aggravate, dyspnœa, œdema, and failure of heart power. Their therapeutic office is to relieve urgent and transitory symptoms.

Dr. T. B. Scott said that in the treatment of arterial hypertension one must remember that there was no exact normal point. One person with a tension of 180 mm. would have no subjective symptoms, while another with the same degree would have vertigo, tinnitus, occipital headache and shortness of breath. It was in these latter cases that treatment was most necessary. Again, one must be very careful rightly to estimate the heart's condition. In most cases a double-barrelled treatment would be of greatest avail—one that would strengthen and brace up the heart's action and at the same time lessen the arterial resistance. He had found in most cases that had not gone on to marked sclerosis and atheroma that strophanthus and the hippurates gave the best and most lasting results. The lowering of tension produced by the hippurates was more gradual, and lasted much longer, than with any other remedy, with the exception, perhaps, of thyroid. They might need to be helped by sod. nitrite. Another remedy that was too seldom used, he thought, was thyroid gland. In stout people especially this acted well, but the heart's action would need very careful watching, and the tension must not be reduced too quickly. This was probably the best and most scientific form of iodine treatment. As suprarenal extract was of organic remedies the raiser of tension, so thyroid was the depresser. At the same time, in many cases of heart failure, even when there was high tension, suprarenal extract was a most valuable heart tonic. The hypodermic injection of liq. adrenalin with 1 or 2 mm. of liq. trinitrin would often pull a desperate case out of danger. Many thought the original cause of sclerosis to be some chronic bacterial poisoning. In support of that view he gave particulars of three cases. Two men and one woman all had chronic bronchial asthma with marked high tension. The men were æt. 65 and 74 and the woman 80 years. All three were treated with autogenous vaccines, and all practically lost their chest trouble. They had no treatment for their tension, nor any medicinal treatment for their bronchitis, but, to his surprise, they all lost their hypertension. Whether this was due to the destruction of poisonous bacteria or to the relief of respiratory strain it was difficult to say, but the results were most encouraging. In conclusion, he wished to say that the ordering of the patients' lives, not only with regard to food and

drink, but also with regard to overwork and worry, was most important.

Dr. WM. ARMSTRONG put in a plea for the old adage that "Prevention is better than cure." His idea was that more attention ought to be paid to the earliest symptoms of arterio-sclerosis. When arterio-sclerosis had thoroughly set in then treatment became a matter of extreme difficulty. Dr. de Havilland Hall, in mentioning suitable health resorts, did not name Buxton. Fifteen years ago he found that when patients were given mineral baths there at a temperature just below blood heat the pulse became much softer. More recently using the sphygmomanometer he found that in a considerable number of cases the baths of Buxton undoubtedly did reduce arterial tension. There was no reason from the composition of the waters, as formerly understood, why they should act in this way; and it was presumed that the cause was the temperature of the water. The discovery of the radio-activity of the Buxton waters, together with the researches of many eminent observers on radium emanation therapy in Germany, opened up further possibilities. In view of the work of Lazarus and Saubermann and others it seemed certain that the radio-activity of waters, such as those of Buxton and Bath in this country and Gastein and Wildbad on the Continent, was a most important factor; and at Buxton he had tried the effect of largely increasing the radio-activity of the waters by reinforcement. The results had been remarkably good. The *strong* radio-active waters greatly increased the output of the body toxins, enhanced the toxicity of the sweat, the elimination of poisons by the kidneys, and the excretion of carbon dioxide in the lung exhalation. Lazarus had drawn attention to the fact that in dealing with radio-active water by drinking or inhalation, greater effect was obtained by the addition of oxygen, a very marked feature being the improvement in the viscosity of the blood. The blood became much more fluid, and the importance of that was emphasised by the remarks just made by Sir Lauder Brunton. In Buxton they added to their mineral water baths in suitable cases oxygen in large quantities. This was tried at first under pressure, but they got no effect; very marked results are now obtained by the addition of nascent oxygen formed by the mixture of perborate of soda and borate of manganese with the radio-active water. Sarason and Winternitz had shown that excellent effects could be obtained in cases of high tension by means of oxygen baths alone without any radio-active water whatever, but the combination was much more effective. The improvement was said to be due to dilatation of the vessels of the muscles, and to the known fact that the increased activity of the peripheral heart sustained the central heart. The question of exercise had been raised in the course of the discussion. For the last twelve months the speaker had been using Bergonie's method, by which exercise was obtained without fatigue. It was a painless method of strong and rapid electrical contraction of the muscles, which were weighted to a considerable extent; he found this method most useful in getting rid of the products of auto-intoxication. He believed that Bergonie's system was of the very greatest value, and that exercise given in this way would do a great amount of good to the threatened sclerotic. Among drugs calomel had been their sheet anchor; but he had got good results with eustenine, which was well taken by many patients who could not tolerate iodide of potassium.

Dr. O. K. WILLIAMSON said that the treatment of high arterial blood-pressure practically included the treatment of arterio-sclerosis, and certainly included that of pre-sclerosis. At the risk of trespassing outside the boundaries of the field of discussion, he would like to say a few words about diagnosis by means of a blood-pressure instrument. One might meet with a case in which, owing to spasm (perhaps local) of the arterial wall at the site of compression, one got a high reading with the instrument when the actual blood-pressure was certainly not above normal. He would therefore urge in all cases the use of the finger as well as the instrument in the examination of the pulse and artery. It was not wise to rely solely upon the instrument. This was a matter of no little importance, and might make the difference between the treatment being effective and being worse than useless. Consider the case of a man who had symptoms suggestive of cerebral thrombosis, and in which the blood-pressure instrument gave a high reading or one somewhat above normal. If they followed what the blood-pressure instrument told them, without using their common-sense and making a digital examination, they would adopt

depressant treatment, with perhaps disastrous results. The moral was obvious. In such a case they must make the blood-pressure instrument their servant and not themselves be slaves to it. With regard to the drug treatment of high arterial pressures, an occasional calomel purge was a very important point. Iodide of potassium, regularly given, was one of the most valuable drugs at their disposal. In cases where iodide of potassium was not well tolerated he had obtained good results from iodipin, as well as from the organic iodide preparation sajodin, especially the latter in doses of 7½ gr., given in tabloid form. Of the vaso-dilators he had found erythrol tetranitrate the most useful. There was a drug which had not received as extensive a trial from British physicians as it perhaps deserved, namely, the preparation of mistletoe known as quipsine. This drug he had found useful in some cases. He had obtained good results by giving it in pill form, in 5 cgrm. doses in amounts varying from six to twenty-five in the twenty-four hours.

Dr. ALFRED MANTLE said that the few remarks he had to make almost amounted to a warning. All of them would be aware how in the eighties, when the influenza epidemic prevailed throughout Europe, the injudicious use of the various coal-tar products became a positive danger, bringing down the high temperature, probably at the expense of the heart. In his own practice at Harrogate every year some cases were sent to him for reduction of blood-pressure. It seemed to him that certain possessors of a sphygmomanometer, directly they saw the mercury rising and approaching 200 mm. Hg. or more, thought it must be reduced at all costs. They forgot that with the gradual increase of pressure there was a readjustment of the cardio-vascular physiology of the patient, and to take means to reduce that pressure was in some cases a wrong treatment. He remembered a well-developed gentleman who had stroked his University Boat for three successive years being sent to him three years ago for reduction of his blood-pressure. He had had previously large doses of iodide of potassium given over a long period, and other vaso-dilator treatment as well. When the speaker examined him he found the heart dilated and very arrhythmic and the legs somewhat œdematous, and in view of all the circumstances of the case he felt it to be his duty not to lower that pressure, which was then 180, but to raise it. He was given nux vomica and strophanthus and he had Nauheim baths and Schott exercises, and after three weeks' treatment the blood-pressure had risen to 200 mm. Hg. and the patient walked better—and with less dyspnœa. His doctor continued the strophanthus for some months on his return home, and when he visited Harrogate again last year the pressure was still 200, but his condition of health was very much improved. Always a great walker, he could now walk three or four miles a day quite comfortably without shortness of breath. It was difficult to know when such patients had got sclerosis in the early stages, and the great thing to do was to catch the case in the pre-sclerotic stage if possible. A very hard-working country doctor, æt. 57 years, was sent to Harrogate for treatment with symptoms of great giddiness when in the vertical position. He was supposed to have disease of the cerebral vessels. His blood-pressure had not been taken, and when I took it was found to be 220 mm. Hg., and the heart's apex was one and a half inches outside the nipple line. There was no albuminuria. He was a man who had taken a good deal of alcohol until five years previously, when he gave it up, and consumed instead large quantities of red meat, deeming this to be necessary in order to keep up his strength for the work he had to do. He was made to rest, and put upon a simple strict diet of milk and fish and calomel and saline purgatives, and by that means the blood-pressure gradually came down, and before he left at the end of four months it was no higher than 150. He visited Harrogate again last year, when it was found to be only 140, and the heart's apex was in the normal position, and he said he could do as hard a day's work as he had ever done in his life. Evidently this was a case caught in the pre-sclerotic stage, and was one of "hyperiesis" described by Sir Clifford Allbutt. Personally, the speaker found that cases did best when treated through the alimentary canal, and he felt sure that, learning as they did from the pathologist that it was the visceral vessels—the splanchnic arteries—which were the earliest affected with sclerosis in the majority of these cases that it was there they had the greatest pressure, accounting for their thickening. In treatment, then, he would say that it was the relief of the abdominal viscera which they ought to aim at by the judicious administration of calomel and saline purgatives, not omitting

to place the patient on a strict diet and enforcing regular exercise.

Dr. C. O. HAWTHORNE criticised the use of the term "arterio-sclerosis" for purposes of clinical description. The term, he understood, was applied by pathologists to a certain definite histological change occurring in the arterial wall, and clinically this condition cannot be distinguished from other forms of arterial hardening. Hence it would seem to be much better in clinical descriptions to speak of "arterial degeneration" or to describe the facts regarded as evidence of such degeneration. To speak of all hardening of the arterial wall as "arterio-sclerosis" is no more justifiable than to describe all such conditions as "atheroma." Similarly, to record the index of the sphygmometer in terms of blood-pressure is open to criticism, as there is at least a doubt whether such index is due to blood-pressure, or at least solely to blood-pressure; and, in any event, the figure registered only one aspect of blood-pressure, namely the height to which it rises. Another, and possibly equally important, aspect of blood-pressure is the time during which it endures, and on this point the sphygmometer gives no information. If this information were desired in a graphic form it could be obtained only by means of the sphygmograph.

In reference to treatment, he found some difficulty in deciding whether the previous speakers, in commending various remedial measures, spoke of the effects of these on the arterial degeneration or on the sphygmometric measurement. It was difficult to believe that any of the remedies mentioned could really modify structural changes which had occurred in the walls of the arteries, and after many observations he (Dr. Hawthorne) was satisfied that neither potassium iodide, nor calomel, nor saline purgatives, nor guipsine had any certain effect on sphygmometric readings. Nitrites, it is true, causes a fall in the reading, but this is of very brief duration. The outcome of the discussion seemed to be that the treatment of arterial degeneration remained exactly what it was before the advent of the sphygmomanometer.

Dr. PRICE: I shall confine my remarks to the treatment of increased blood-pressure, and for the most part they will be based upon investigations which I have conducted in the wards of the Mount Vernon Hospital. First of all, it seems to me we should be cautious in arriving at the conclusion that a blood-pressure outside what is supposed to be the normal limits necessarily indicates a diseased state. Some people are perfectly healthy with a pulse of 60, and others with a pulse of 80. May not the same principle hold good in regard to the blood-pressure? If that be so, we should make inquiries in regard to two points: (1) What was the patient's usual blood-pressure previously—in other words, is there a persistent and decided rise from what we know was the patient's usual blood-pressure? and (2) are there any other symptoms or physical signs than a supposed abnormal blood-pressure, such as headache, shortness of breath or other indications of cardiac failure, or of renal disease. That is, we should be careful not to draw hard-and-fast conclusions from blood-pressure readings alone.

If I may be permitted to say so, I think we should exercise great caution in forming opinions regarding the results of any remedial measures which we employ. There is a great danger of our conclusions being unwarranted when they are based upon merely general observations. It seems to me that it is at least possible that a good deal of what is commonly believed in regard to the treatment of supernormal blood-pressure does not rest upon absolutely reliable or proved data. This applies particularly to the matter of the administration of drugs. It should never be forgotten that most of our beliefs in regard to the actions of drugs rest only upon experiments on animals, and animals which are in normal health. Now, although it may be proved that particular drugs have certain actions on healthy animals, it does not necessarily follow that they have the same actions on human beings with diseased conditions.

In trying to arrive at facts I adopted the following precautions in my investigations. I mention them here because I think that if these precautions were always exercised we should not be so liable to come to unjustifiable conclusions regarding the results of the therapeutic measures which we employ.

The blood-pressure measurements were taken either by myself or on nearly every occasion by one particular individual, with whose results I repeatedly compared my own and always found them to be the same. Observations were made in each case over a prolonged period, in some cases up to several months. The measurements were taken at exactly the same hour of the day, at the

same time in reference to meals, and under precisely the same conditions (that is, the patients were kept in bed, etc.). I employed Martin's modification of the Riva-Rocci apparatus, and the same instrument was used on each occasion. The readings were limited to those of the systolic pressure, because I am still doubtful whether we yet have a method of accurately determining the diastolic pressure. Also, I confined my investigations to cases with a regular heart action or with only an occasional irregularity; because in other cases, where there is a constant variation in the strength of the pulse-beats, it appears to me that it is not possible to obtain accurate measurements.

Let me now mention some of the results of my investigations. First of all, one important point was elicited which I think has a very important bearing upon the subject of our discussion this evening, and I may say that the conclusion arrived at is based upon a very large number of observations. It is this: that in some cases I found considerable fluctuations in the readings from day to day. I am not now referring to the variations associated with meals, physical exertion, or mental states, but to diurnal variations which appear to be quite independent of these. Let me just mention one case, namely, that of a middle-aged man, suffering from a moderate degree of thickening of the vessel wall and a slightly enlarged heart, but with no evidence of kidney disease, who was under my observation for five months, and whose blood-pressure measurements I took myself at the same time of the day nearly every day and under precisely the same conditions for many weeks. I found diurnal variations up to 26 mm. of mercury. Now, if in by no means a small proportion of cases there are considerable normal fluctuations from day to day, it shows how careful we should be in coming to conclusions in regard to the action of drugs on the blood-pressure in man. It should always be remembered that any changes observed after the administration of a drug in disease may be due to the natural course of the malady.

Another point which we should bear in mind is that there are variations in the blood-pressure under physiological conditions, such as meals, physical exertion, and mental states.

The result of my investigations may be summarised as follows:

In regard to the treatment of severe degrees of increased blood-pressure, with the exception of venesection as a purely temporary measure, I was very disappointed, and especially with the results of the administration of the so-called appressor drugs. I found these drugs valueless in almost every case, not only in regard to their permanent, but also in regard to their temporary, effects. As examples I may mention that I tried erythrol tetranitrate in large doses, samples of gauranteed pure sodium nitrite (obtained from Mr. Martindale) up to 10-gr. doses, iodide of potassium up to 1-drm. doses, and nitro-glycerine up to $\frac{1}{50}$-gr. doses, each three times a day. In many cases I took measurements every few minutes for several hours after individual doses.

In regard to slight and moderate degrees of increased blood-pressure, I think much can be done in a considerable proportion of cases but not in all.

In respect to the comparative value of the various therapeutic agencies, better results were found from other forms of treatment than that of drugs. Among the former I would put rest, exercise, diet, and free elimination. In my opinion, rest, both physical and mental, is of paramount importance, and its value is not sufficiently realised. It is sometimes necessary to keep the patient in bed, for varying periods according to the nature of the case. In regard to diet, I think we should pursue a middle course, both in regard to the quantity and quality of the food. Exercise should be regular and moderate, and great discrimination on the part of the physician is required. Purgatives are usually of distinct value. I tried venesection, up to 30 oz. In one case the blood-pressure fell about 30 mm., but began to rise again in the course of a few days. The withdrawal of blood is sometimes of a very great value as a purely temporary measure. Even in cases of slight supernormal blood-pressure the results of the administration of drugs were by no means infrequently negative. For example, in one case I gave a large dose of erythrol tetranitrate, and took measurements every ten minutes for three hours; the result was negative. This was a surprise to me. In no case was the administration of iodides followed by a reduction of blood-pressure; yet in some cases the drug seemed to be of value in some other way.

In conclusion, it appears to me that there is much need for further clinical investigations in regard to this subject.

Dr. WALTER BROADBENT said that nobody in the course of the discussion had mentioned the factor of heredity. The fact was, however, that certain people were born with low tension. They drank, and ate, and smoked as much as they liked. At the present moment he knew an old

gentleman, who had led what would be called a "fast" life, and who still smoked he thought about three cigars a day, and generally had a liberal allowance of champagne every night. And still, at the age of eighty-five, had quite a soft artery. His father and mother both lived to be over ninety; and he believed that whatever that man did he could not raise his blood-pressure. Another class of people, unfortunately, were born with high tension. A fellow student of his was rather proud when at college of showing the sphygmographic tracings of his pulse as typical of high tension. He died at the age of forty of apoplexy. He was quite a steady man in college; he did not know what he might have been afterwards. There were a very large number of hereditary cases which the physician could not alter, but in between these two groups of hereditary cases there were a number of people who had a more or less moderate blood-pressure, and these could be influenced. He did not quite agree with Dr. Luff on the question of golf. In a great many cases of real arterio-sclerosis the game did a great amount of good. It did keep down the tension. Of course judgment was necessary. One would not put a man with an aneurysm on to playing golf—an instance of which had come under his notice at Brighton. Another point which had not been brought out that evening was a thing which had struck him on examining the condition of the arteries in a large number of out-patients. A few years ago it was his custom to make a note of the condition of the arteries in the case of every out-patient, and he got notes of some two thousand. He found that among the men and women below fifty having arterio-sclerosis the percentage of the men was about ten times that of the women. But between the ages of fifty and sixty the women began to catch up very quickly on the men, and between the ages of sixty and seventy, while there were 85 per cent. of such cases among the men, the very close approximation of 78 per cent. was recorded among the women. It seemed probable that in the case of women before the menopause the monthly blood-letting was a means of keeping down the pressure, and this rather pointed to the benefits of blood-letting. He entirely agreed with Dr. Williamson that to feel the pulse was more valuable than to take a sphygmomanometric reading. When high pressures were recorded a good many people were needlessly frightened, although actually the amount of high tension was not grave. He had tested guipsine very carefully, but it had proved of no value in his hands, and he had given up its use.

Dr. WALTER CARR said that he was reminded of the discussion held twelve months ago on "The Sphygmomanometer in General Practice"—a discussion to which that evening's debate was the natural sequel. He learned many things on the former occasion, but nothing impressed him so much as the remarks of Dr. Harold des Voeux, who referred to the immense amount of harm done by the introduction of the sphygmomanometer, which had converted many more or less healthy people into chronic invalids. He had since then realised from his own experience that it was easy in this way to induce invalidism and possibly little more than a mere vegetative existence among people who might otherwise be leading fairly useful and energetic lives. Their lives might perhaps be protracted, but they were of very little use to anyone, and whether or not they had high tension in their vessels they certainly had it on their brains. Of course they must all agree with Dr. Armstrong that prevention was better than cure, but the unfortunate thing was that so often they could not get these patients in the early stage. Usually they did not come under observation until they had been suffering from high tension for a considerable time.

In treatment it was necessary to recognise the essential fact that high tension was a form of compensation. In the case of a hypertrophied left ventricle they did not regard it as the great object of their treatment to get rid of the hypertrophy, for this was the necessary result of antecedent changes, and although it involved dangers of its own, yet any interference with it might lead to a speedy termination of the patient's life. The same was true of high tension. They were often quite unable to reduce it permanently, and even could they do so they might frequently do the patient more harm than good. As had already been pointed out that evening, the whole mechanism of the circulation was altered in these cases, and frequently, if the tension were lowered, the patient felt miserable, and only became happy again when it had returned to its previous high level. The essential thing was to recognise and treat as far as possible the cause of the high tension, realising that apart from heredity, advancing age and kidney disease, it was a result mainly of what the late Sir Andrew Clark called a departure from the ways of physiological righteousness. The patient must be induced to lead as temperate and quiet a life as possible, and to practise moderation in all things. Reference had been made to the importance of walking in bringing down high tension. As a pertinacious and persistent pedestrian he was glad to hear this, but he should like to add that there was a great difference between walking on the level and walking up hill. Walking on the level was monotonous and much less beneficial, but hill-climbing, suitably graduated, was, as they had learned from Sir Clifford Allbutt, by far the most valuable form of walking, and one which was much more to be encouraged than mere walking on the flat.

Dr. F. DE HAVILLAND HALL, in replying on the discussion, said that he was keenly interested in

Sir Lauder Brunton's remarks on the viscosity of the blood, and the good effect of oxygen in these cases. He himself in the past had at times been unable to get any blood on puncture. As to what Sir Lauder had said about old-fashioned remedies, in his early life he knew a man who was in the habit of taking 3 grains of Plummer's pill every night for a number of years. He was a man who lived a luxurious life, a very large eater, and took hardly any exercise. By taking more exercise under medical advice he was able to dispense with the pill. He lived for thirty years afterwards, and died at the age of seventy-seven. He agreed with Dr. Luff that arterio-sclerosis might be met with accompanied by low tension. In some cases the pressure was undoubtedly low. But he could not agree with what he had said about tobacco and alcohol. He was convinced that these were important factors in the causation of arterio-sclerosis. He could recall a case of a man of seventy—a very distinguished man—who had lived an active life. One night he had a severe attack of angina, and on seeing him next day the speaker found him to be suffering great pain and in a state of much anxiety about his condition. He was a very great smoker, consuming six or eight cigars per day, and for a man over seventy that was certainly injurious. He was cured of the tobacco habit almost entirely, and on giving it up his arterial tension fell, and there was no recurrence of his anginal symptoms. He was quite certain that tobacco had a great effect in the causation of this condition. As to golf, this might be of benefit, although on this question he had spoken tentatively. He was interested in what Dr. Luff had said about Sidmouth and Torquay, because these were seaside places. Other seaside places—he was sorry, Dr. Walter Broadbent being there, to have to include Brighton—were not so favourable. "Dr. Brighton" was useful for many things, but not for high tension, and disastrous results had happened in some cases through being sent there. He agreed with Dr. Bezley Thorne as to the extreme importance of attending to the alimentary canal. That was one of the most fertile causes of the toxæmia which produced arterio-sclerosis. Dr. Bezley Thorne made some interesting remarks about baths, and he was hoping that he would have developed that part of the subject rather more, and indeed for that reason had not touched upon it to any great extent himself. He was very glad Dr. Scott, of Bournemouth, had spoken, as it was from his excellent paper in the 'Practitioner' last year that he derived his own information as to the value of the hippurates. He also quite agreed as to the great value of thyroid gland in some of these cases, especially in the obese. A small dose of 2½ gr. daily would effect a great deal in these cases, especially at the menopause. He was suggesting before the meeting that in cases of this sort one might try what vaccines would do. He was at one with Dr. Armstrong in what he had said about prevention. He was sorry he had not mentioned Buxton. He had sent patients to Buxton from time to time, but not cases of arterio-sclerosis, and therefore he could not speak with authority. From what Dr. Armstrong had told them about the radio-activity of the Buxton water, one would certainly be inclined to try the effect of Buxton. With Dr. Williamson he was in complete accord as to the extreme importance of recognising what was the condition of the pulse in some cerebral cases. He was called down to a patient in the country supposed to be suffering from cerebral hæmorrhage, and found him with very low tension and cold extremities. This was not a case of cerebral hæmorrhage but of thrombosis. He ordered him some alcohol and nux vomica, and in the course of a week or ten days he was much improved and afterwards was able to resume his work. It was important to be quite certain that one was not dealing with thrombosis instead of hæmorrhage. Dr. F. W. Price, he thought, took rather a pessimistic view of things. But he was disagreeing with the results of clinical experience. They did find these drugs answer, and he was disposed to use them, even though distinguished men in the laboratories found that they did not come out exactly as they would expect. He thought Dr. Hawthorne was somewhat meticulous. They must clinically assume something. "Arterio-sclerosis" was a convenient expression, and he would stick to it, even at the bedside. They were pretty well agreed as to what it meant. As to the lack of effect on sphygmomanometric readings from drugs, he did not advise large doses, but he did believe in the continued administration of small doses of iodide of potassium. He recollected one case in particular, the patient having been under observation for six years, in which he was convinced that the steady administration of a small dose had been distinctly beneficial. He ought to have mentioned in his paper the point about heredity, to which Dr. Walter Broadbent had alluded. Some people were born with hard arteries, and others with soft. He himself was born with soft arteries, and he recollected feeling his father's pulse when his father was in his eighty-ninth year, and it proved to be quite soft. Dr. Carr had spoken of the very straight observations made by Dr. des Voeux on the previous occasion, and he had hoped to secure his attendance there that evening, when doubtless they would have listened to some more scathing remarks. But in connection with this aspect of the subject, he often thought of and sometimes quoted the old Latin proverb, meaning "People sometimes lose life by the means they take to save it."

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THE TREND OF THOUGHT IN RECENT PHARMACOLOGICAL RESEARCH.*

By SIR WILLIAM WHITLA, M.A., M.D., LL.D.,
Professor of Materia Medica and Therapeutics, Queen's University, Belfast.

THE orator, after a *résumé* of the history of former therapeutic progress, proceeded to point out that the advances made in one era can often be traced backwards to their origin in a rational or accidental discovery made at a much earlier date, and sometimes to a research, the importance of which was overlooked at the time. Such a starting-point may be found in the epoch-marking researches on the action of methyl-strychnine, conducted by Crum Brown and Fraser in 1868. It was well known that ammonia and its derivatives, when administered to animals, caused stimulation of the spinal cord and medulla, which ended in convulsions; it was also recognised that the chemical constitution of the vegetable alkaloids corresponded to the type of an ammonia derivative. These experimentalists made the discovery that by linking the organic alkyl radical, methyl (CH_3), to the nitrogen of the complex molecule of strychnine, the resulting comparatively slight change in chemical structure altered in a remarkable manner the physiological or pharmacological action of the alkaloid. The alteration was manifested by a change from the convulsant effect on the cells of the cord to a paralytic one on the terminals of the motor nerves, such as is witnessed in the well-known symptoms of poisoning by curare. This change of physiological action being manifestly due to the transformation of the original tertiary base into a quaternary ammonium compound, it was not to be wondered at that almost identical results were

* Abstract of the Annual Oration at the Medical Society of London.

obtained by altering in the same way the chemical constitution or structure of a number of the other alkaloids exercising widely different actions on the human body.

Similar change in physiological action is also obtainable by linking other alkyl radicals, as C_2H_5 instead of CH_3, the radicals being, in each case, previously combined with iodine or bromine. That this change in action is not a mere coincidence or accident is demonstrable in a remarkable manner by the methylation of curare itself—the drug which, in its natural state, affords the most typical example of a paralytic effect upon the motor nerve-endings. This substance contains two ammonia bases—the active curarine and the comparatively inert curine. Now, if the latter base be altered by the before-mentioned manipulation with methyl, the transformation in its chemical constitution causes it to become about 230 times more poisonous than before, and the resulting new compound is found to be identical with curarine, the active principle of the drug.

Sir Lauder Brunton, speaking, in 1886, of Crum Brown and Fraser's discovery as directing pharmacological advance along a new path, prophesied that another twenty years would not only add greatly to our stock of remedial agents, but to a better knowledge of our patient's condition, ushering in the era when we should be able to ensure the drug action we desired without feeling that our patients often died for lack of knowledge, not on our part but on that of our art. We all feel painfully that such a millennium is still far off, but, nevertheless, we are steadily, if slowly, approaching it. If we interpret Brunton's forecast concerning "our better knowledge of the patient's condition" as implying a clearer conception of the incessant changes taking place in the protoplasm of the body cells, it must be conceded that enormous strides have been made in our progress in this direction, since, during the last quarter of a century, a chemical basis has been industriously constructed, upon which the science of pathology now securely rests—a fact not sufficiently recognised, even by pharmacologists.

It was expected by pharmacologists that Brown and Fraser's discovery would immediately revolutionise therapeutics, but in this they were soon disappointed. The application of a few swiftly constructed generalisations to the solution of intricate therapeutic problems at first only led to failure and chaos, and, as always happens, a revolt against the new doctrine soon appeared. Even at the present hour some pharmacologists labour under the old incubus of trying to explain the action of drugs on the theory of some purely physical or mechanical property in their atoms or molecules, whilst others are falling back hopelessly upon the ancient vitalistic conception. Hence arises the importance of a scrutiny of our present position, since the problem is not one to be decided only in the laboratory, but to be finally tested by the practitioner at the bedside.

In examining the theory of the relations between pharmacological action and chemical constitution we must put aside the question of the mere chemical composition of a drug. It is not the number or nature of the atoms in a compound which we have to think of, but the plan of structure by which these are built up and linked to each other in the drug. In other words, it is not a consideration of the number or nature of the individual bricks or building stones employed, but the manner in which these are grouped in the molecules of the compound radicals determining the final chemical construction or constitution of the compound. This vital distinction between the structure and composition of a chemical compound is seen in the empiric formula $C_6H_{12}O_4$, which is the label attached to no fewer than sixty-six compounds—all totally different substances. It is true that a simple change in the mere composition of a salt may change or vary greatly its action, but this will generally be found to be due to some alteration in its solubility which causes retardation of its absorption or acceleration of its elimination. Thus the victim of the malarial parasite can be often speedily rescued from his perilous toxæmia by a moderate dose of the hydrochloride of quinine, when he would perish with an enormously larger amount of the insoluble alkaloid or its sulphate lying unabsorbed in his gastric cavity.

After the demonstration of the results of methylating the strychnine molecule came the valuable generalisation of Brunton and Cash on the actions of the carbon compounds. They pointed out that the two great fundamental groups of organic chemistry differed materially in their pharmacological actions. The substances built up on the open chain plan, the type of which is marsh gas

(CH_4) and chloroform ($CHCl_3$), all paralyse the nerve-centres and tend to exert an anæsthetic action, whilst the close chain or ring series, represented by the benzine nucleus, produce convulsions or spasms before paralysing. Liebreich, seizing the truth of this common paralytic action of the open chain or fatty series of the carbon compounds, started out in search of a new hypnotic, which ended in the discovery of chloral, as Schmiedeberg likewise gave to therapy the invaluable hypnotic—paraldehyde.

When one of the hydrogen atoms in the closed benzene ring is replaced by the radical hydroxyl (HO) phenol is formed, and as each of the other hydrogen atoms is substituted by a further hydroxyl addition, or by methyl, the antiseptic power of the resulting aromatic compound proportionally increases. It was found that by varying the position of hydroxyl in the ring valuable antipyretic qualities could be developed; thus was achieved a notable triumph in therapeutics by advancing from hydroquinone to the synthetical building up of kairin, thallin, and finally of antipyrine.

Since then innumerable examples have accumulated equally suggestive of this close relation. It is noteworthy that these examples tell in opposite directions. Not only is the synthetical chemist able, by altering the structure of the molecule, to intensify greatly the action of the compound; but he can, with equal ease, render the poisonous drug inert by introducing into the molecule such acidic groups as the sulphuric, sulphonic, and carboxylic. Thus the poisonous phenol is converted into the inert phenyl-sulphuric acid, and morphine-sulphuric acid can be awallowed with impunity in drachm doses. Notwithstanding, however, these accumulating instances of close relationship, it is still impossible for the pharmacologist to foreshadow with certainty the physiological or therapeutical action of a particular untried drug, so that rational must proceed in company with empirical therapy. But the shrewd guesses of a couple of decades ago are now being replaced by the stronger probabilities of later years, and by the almost certainties of 1913. It is often argued by the sceptic, with an apparent show of reason, that this relation between chemical structure and pharmacological effect, if it be a definite law, should afford the certainty of a mathematically correct prophecy. That it does not, is due to the fact of our want of knowledge about the construction of the molecules of the highly complex carbon compounds, whose number almost suggests infinity, and of which but few comparatively have been studied structurally. Each discovery of the true nature of the graphic formula of these shows that Nature is gradually, if reluctantly, yielding up her secrets to the chemist, who at once seizes upon the advance as a new stepping-stone from which to approach the creation of a new compound with the almost certainty of obtaining a specific action upon some particular type of body cell or tissue. It is true that unforeseen secondary actions of the newly created drug may vitiate the desired result and prove a barrier to its safe use, but often a further study of its complex molecule suggests a new substitution process by which untoward effects can be eliminated.

No stronger proof could be produced for the demonstration of a truth in medicine or in any other science than the long list of new synthetic antiseptics, antipyretics, analgesics, local anæsthetics, and hypnotics, the discovery of which is due to the theory of this relation. If we even set aside the entire series of new remedial agents just mentioned, salvarsan would alone demonstrate the truth of the contention. Ehrlich, in the introduction to "The Experimental Chemotherapy of Spirilloses," states that from the very beginning of his medical studies he became possessed with the conviction that "If a connection between the constitution and the physiological action were granted, this must be pushed further and the tissues must also participate in the distribution, and whereas formerly the substances were offered to the medical man by the chemist for testing purposes, the conditions could now be reversed and the chemotherapeutist could give the chemist points which lead to the desired recovery of genuine curative substances."

The question of whether or not Ehrlich has discovered a cure for syphilis sinks into insignificance when contrasted with the importance of the fact that his methods are based upon such a rock-bed of truth as must ultimately revolutionise the treatment of all parasitic diseases by chemical agents. That no single step in his laborious search for a remedy against the spirochætes was taken empirically or at haphazard is evident from his repeated assertion that from start to finish every advance was made in a most purposive manner and only after an exhaustive study of the molecular con-

struction of each of his "606" tested compounds. How far Ehrlich pushed his conviction beyond the theory of mere relationship brings me to the consideration of the second great fundamental principle underlying the conception of a purely chemical basis for all therapy.

His term "chemotherapy" implies a great deal more than the acceptance of an established dependance of action on chemical constitution. With this new word another working hypothesis leaps into view, and it is essentially different from the one which we have been considering. The mixing up of these two theories has already threatened a check to therapeutic advance. Stated in its most advanced form, this second theory attempts to explain the action of a drug as the result of a chemical combination of some of its groups with other constituent groups or radicals in the complex molecule of the living protoplasm of the cell or tissue upon which it acts. It must be at once conceded that such chemical reaction can only be proven in a few cases, and therefore does not rest on the solid basis of the established relations between structure and drug action.

Let us put this working hypothesis of chemical reaction to the same severe test as we have applied to the outcome of Brown and Fraser's discovery. And here let me remind you that the molecular theory upon which chemistry is based is still but a working hypothesis, and probably must remain as such for all time. It has long been known that many drugs suffer a fundamental change between the periods of their absorption and final elimination from the body. The direction of this change, as put by May, is usually in the direction of a transformation of an active or poisonous substance into a less harmful compound, and though it cannot be accepted as proof that the effect of the original drug is due to a chemical reaction between its molecules and those of the living protoplasm on which it acts, it may nevertheless be incidentally mentioned that this knowledge has been put to practical account by suggesting to the synthetic chemist the planning of a new compound on the same type as the transformed eliminated product. In this way, from a study of the behaviour of anilin in the body, phenacetin and other valuable para-amino-phenol derivatives have been added to the therapeutic armamentarium.

The great difficulty of demonstrating the doctrine of a purely chemical basis for pharmacological action rests in our incomplete knowledge of the chemical construction of the living cell protoplasm and of the physico-chemical processes incessantly occurring therein. The puzzling and highly complex constitution of the proteid molecule is gradually being cleared up, and already about a score of its cleft-off building stones have been examined and proved to be of definite chemical composition and constitution, containing strong basic and acid groups capable of combining with each other to form an infinite number of compounds. Though we are still a long way from a synthetical construction of a proteid molecule, the building up of the polypeptids by Emil Fischer strongly encourages the hope that this giant achievement is a possibility in the not distant future How vast is the field for such research, and how numerous are the amino-acid groups all capable of combining with each other through their basic and acid radicals may be dimly appreciated by the estimation of the molecular weight of hæmoglobin as 16,669.

These considerations clear the way for a glance at Ehrlich's great side-chain hypothesis or working theory advanced in order to explain the phenomena of the action of the antitoxins of diphtheria and tetanus. It is based entirely upon the chemical conception, and was built up to explain the action of the most complex organic compounds known to science in order to elucidate the phenomena of immunisation. It recognises the fact that the toxin molecule contains a large number of complex organic groups or radicals bound together to a central chain or nucleus.

Some of these radical groupings possess a chemical affinity for other complex groups existing in the protoplasm of the cells susceptible to the poison. These chemical affinities constitute in the language of Ehrlich the side-chains known as the anchoring group or haptophore, which combines with a receptor radical in the cell protoplasm, and thus fixes or anchors the toxin molecule to the protoplasmic molecule. The harmful radical in the toxin, being thus fastened in close relationship with a chemical group in the cell, combines with it through its toxophore radical, the chemical reaction accomplishing the injury or death of the cell.

According to this hypothesis no result will

follow the injection of a toxin unless the cells of the organism contain in their molecule a group or radical, known as the receptor, which possesses an affinity for the haptophore or anchoring radical in the toxin as well as a group capable of being injured by the toxophore side chain of the poison. The phenomenon of natural insusceptibility is thus easily explained since the immune animal contains in its cellular protoplasm no receptors for the fixation of the toxin molecule. Of much more practical importance to the therapeutist is the working out of this hypothesis when applied to the elucidation of acquired insusceptibility or immunisation, and of still greater importance is it in solving the problem of the action of antitoxins in the cure of disease. When doses of the toxin short of producing a fatal result are injected the chemical reactions just described are assumed to have occurred, but the smaller amounts, instead of destroying the cell, only serve to excite it to regenerate its receptors. The cell is also further stimulated to produce more of the chemical receptor substance than is merely required to replace that which has already combined with the toxin, the excess being showered into the blood constituting the antibodies found in the serum. These, therefore, are to be considered as free cell receptors with unsaturated chemical affinities which immediately combine with the haptophore radicals of the toxin, thus effectually preventing them anchoring to the cell protoplasm, without which fixation a toxic effect cannot be produced.

Serum therapy has been often confused with vaccine therapy to the retardation of therapeutic advance. I have used the term "toxin" only in its now scientifically restricted sense as the poisonous product of a true *secretion* of a bacillus. Probably only four pathogenic bacilli secrete a chemical poison—those of diphtheria, tetanus and botulismus, and *pyocyaneus* bacilli. All the other vegetable parasites contain their poisons as an intra-cellular chemical or endotoxin, which can only operate deleteriously on the tissues after the death and dissolution of the microbe. This fact constitutes the fundamental difference between serum and vaccine therapy. Only toxins lead to the formation of true antibodies in the serum. When we remember that Behring was able to immunise an animal so that one thousandth part of a drop of its serum was sufficient to neutralise a lethal dose of the chemical toxin, we realise that there is nothing in the whole realm of therapy upon a more secure foundation than the serum treatment of diphtheria and tetanus. The comparative failure of the antitoxin of the latter disease only strengthens the structure upon which the therapy is erected, since by the time the first symptoms of trismus have appeared, a chemical combination between the groups of the toxin and the molecule of the cell protoplasm has already taken place. We are even justified in asserting that if we could know the exact moment at which the bacilli of these diseases enter the body, by an immediate resort to the serum a fatal issue would always be averted.

It is necessary to realise the truth of this in order to judge rightly of the value of Ehrlich's hypothesis, which explains, on a purely chemical basis, almost every problem met with in the marvellous processes of active and passive immunisation, notwithstanding the theoretical qualifying views of Arrhenius and Madsen regarding dissociation, or the adsorption theory of Bordet and Biltz and the recent experiments of Abderhalden with the polariscope. But even if we assume that the side-chain hypothesis does not assist us in establishing *proof* that the action of the antitoxic serum is of the nature of a true chemical reaction, the following facts must be accepted as conclusive evidence that the result is due to the operation of laws which govern all other chemical reactions:

(1) The neutralisation or saturation of the toxin by the antitoxin occurs in the test-tube as well as in the animal body.

(2) It is more rapidly effected by mixing together strong solutions than weak ones.

(3) It occurs more quickly under warmth and is proportionally retarded by cold.

(4) A very marked elevation of temperature always accompanies the reaction *in vitro*, the degree of heat having been accurately measured by Arrhenius and Madsen, who found it to correspond to about half the elevation which follows the neutralisation of a strong acid by an active alkali.

(5) The toxin and antitoxin do not perish, or are not destroyed by the chemical combination; though the former is deprived of its injurious property for the time it can afterwards be recovered from the solution.

(6) The neutralisation of the toxin has been

demonstrated to proceed in obedience to the law of constant proportion; that is to say, a given amount of the chemical poison, or multiples of this amount, are always saturated by corresponding multiples of antitoxin—a fact which alone would establish the purely chemical basis of the reaction.

(7) If the tetanus toxin be mixed with an emulsion of the brain of a healthy guinea-pig it becomes innocuous, chemical combination being thus brought about between it and the nerve protoplasm, probably in the exact manner in which this takes place in the living animal after injection of the poison.

A study of the evolutionary steps in the side-chain hypothesis is of considerable interest. It started in Ehrlich's mind on observing the effects of various staining agents on different parts of the animal cell. After studying the chemical structure of the dye molecule he was led to the explanation of the phenomena of immunisation. It is noteworthy that he first denied the possibility of any similarity between the action of a chemical substance of known composition and of an antitoxin. He was, however, inexorably led by his work to the conviction that the protoplasmic molecule possessed chemo-receptors which united with the haptophore or anchoring groups in the drug.

Dixon argues with a brilliant *naïveté* that since these hypothetical receptors are obviously distinct from food receptors it is difficult to regard them teleologically as pegs specially designed for the physician on which to hang his drugs. From a consideration of the results achieved it must be conceded that they are pegs strong enough to support the heavy superstructure of chemotherapy, the coping-stone of which at present is neo-salvarsan.

Led first by the truth of the generalisation arising from the research of Crum Brown and Fraser, Ehrlich proceeded on his path of exploration by applying at each step his side-chain hypothesis to the elucidation of the complex reactions occurring in the human body after the injection of various dye-stuffs and a host of arsenical compounds, with the result that he was enabled to discover an agent possessing the maximum parasite-destroying power with the minimum of organotropic action. But this was not all; the blending of the solution of two problems which confronted him is to be seen when we examine his final achievement. He had not only to solve the problem of killing the parasite without injuring the patient, but he had to cure the disease, which was liable to be aggravated seriously by the rapid disintegration of the slain spirochætes. In his method we find chemo- and sero-therapy working side by side, the one aiding the other. A dose of salvarsan injected into the veins of a syphilitic mother effects the dissolution of the parasites, whose liberated toxin at once leads to the formation of antibodies in the parental blood; these, on being conveyed to the infant in the mammary secretion, destroy its myriad spirochætes or render their propagation impossible, as in the diphtheritic disease, even when no trace of the salvarsan molecule can be detected in the mother's milk.

In establishing chemotherapy Ehrlich's aim was to effect a complete sterilisation of the body by a single full dose of the anti-parasitic drug without injuring the tissues, a result which he designates the *therapia sterilisans magna*. It is remarkable that in both this result and in the chemical combination theory which led to its achievement, he was forestalled by Binz, of Bonn, in a most important research made in 1867, the year previous to Brown and Fraser's discovery. Binz, when experimenting on the action of various drugs on the infusorial organisms met with in ordinary hay infusions, discovered that active paramecia were readily destroyed by a 1 in 10,000 solution of quinine, though they withstood the action of enormously stronger solutions of strychnine and other poisons of vegetable origin.

Three momentous conclusions were drawn from this simple experiment. Firstly, that the cause of malaria, which was then unknown or dimly guessed at as a nervous manifestation, was probably protozoan, since malarial disease was so readily cured by quinine. Secondly, that by administering 15 gr. of the hydrochloride to an average man a solution of twice the strength in the blood necessary to kill amœba was formed, thus fulfilling the ideal of *therapia sterilisans magna* of Ehrlich. Thirdly, at a later period the working hypothesis of true chemotherapy was applied, by which it was inferred that the quinine entered into chemical combination with the protoplasmic molecule of the parasite, whereby it was robbed of its power of absorbing oxygen and died of asphyxia.

From a consideration of the chemical basis of

serum therapy, one is naturally led to test the value of this working hypothesis as affording an explanation of the advances made in vaccine therapy by Wright. We shall pass lightly over these brilliant achievements, not because they are of less importance, but because the progress made in this direction does not very obviously appear from Wright's published writings to have been first started by the impulse of a chemical theory, as was the case in the more apparently purposive scheme of Ehrlich's researches.

The recent Witwatersrand Report of Sir Almroth on "The Treatment of Pneumonia" shows that vaccine treatment owes its origin to the conception of a true chemical combination occurring between the remedial vaccine and the bacterial protoplasm. Thus, he states that there is within the human body a laboratory which can, within certain limits, furnish bacteriotropic substances which will enter into destructive chemical combination with any and every variety of bacterial protoplasm without poisoning the tissues or becoming inert in the blood. All immuno-therapy, and in particular all vaccine therapy, has been built up, he says, "upon these facts and upon the fact that the chemical machinery which elaborates bacteriotropic substances is set in motion whenever a chemical attack is made upon the tissues by dissolved bacterial protoplasm." He objects to the term "chemotherapy" being restricted in its application to specific treatment by drugs, since, in all cases, both in serum and vaccine treatment as well as in specific drug therapy, the attack upon the microbes is a *chemical* attack.

The chemical reactions are much more complicated and hence more difficult of demonstration than in the case of the simpler neutralisation of a toxin by its antitoxin, because, as already stated, no true antibodies are produced during vaccine treatment. Nevertheless the serum of an immunised animal will kill the corresponding bacteria when these are added to it *in vitro*, though in a distinctly different manner from that by which the diphtheritic and tetanus antitoxin act.

Two substances are required for this destruction of the microbe; one, the so-called *immune body*, is produced during the injection treatment, the other, known as the *complement*, exists already in the normal serum and is not increased by the injections. The side-chain doctrine hypothecates the immune body as possessing two receptors, one of which binds it to the bacterial endotoxin, whilst its second affinity satisfies itself by combining with the complement. The possession of this double affinity of the bacteriolysin is recognised in its name of amboceptor. It must be regarded as an intermediary body which (in the chemical language of Wells), on being added to the bacteria during the process of immunisation, gives to them a chemical affinity for the complement existing already in the blood-serum. It is therefore the complement and not the immune body which effects the destruction of the bacteria.

The sceptic may say that this explanation is pure fancy and that chemical laws have nothing to do with the result, but Hektoen has been able to isolate the complement and to prove that it is a definite chemical compound. He has rendered it inactive by causing it to combine with the ions of calcium, magnesia, and sulphuric acid, and Manwaring has succeeded in divorcing it from these ions by a simple chemical reaction. Other investigators have shown that it is a body which, though closely resembling an enzyme and very susceptible to heat, nevertheless combines with the amboceptor, in obedience to the law of definite or multiple proportions which prevails in every true chemical reaction.

The most inscrutable problem arising in serum therapy is the phenomenon of hypersensitiveness which appears on the re-injection of a serum, and causes death when all is apparently going well with the patient. Wolf-Eisner pointed out that the fatal result was due to the operation of a general biological law governing the action of a heterogeneous albumen, viz. that it causes the formation of reactive substances. Recently, this hitherto insoluble crux has been brought under the grasp of the chemical working hypothesis in a most unexpected way by Friedberger, who, by mixing quantitative proportions of antigens, antibodies, and complement obtained from healthy serum, was able to liberate the chemical compound which produces the tragic phenomenon of hypersensitiveness.

From these important discoveries which have established a true working theory for serum therapy, vaccination treatment, and hypersensitiveness, we derive faith that the long cherished hope of dealing effectually with all bacterial diseases will be

soon realised, especially as it has been abundantly proven that the protoplasmic molecules of nearly all the pathogenic organisms possess receptors capable of combining chemically with various staining reagents.

Till quite recently it must be confessed that all the more important advances in therapy left the greater problem of the treatment of the diseases caused by endotoxin microbes untouched. Neither did the triumph of salvarsan aid us directly save by lifting therapeutics out of the old rut of empiricism by demonstrating what could be achieved by the application of rational methods based upon a sound chemical theory.

The fringe of the dark cloud which had overshadowed the therapeutics of the commoner bacterial affections has recently been lifted by the experimental researches of Morgenroth and his co-workers. Writing of these results, Wright states that "they are destined to stand out as a landmark in the history of pharmacotherapy because they furnish the first demonstration of the possibility of preventing and curing a bacterial—as distinguished from a protozoal or spirochætal—infection by the administration of a drug." The experiments were made by injecting mice with the pneumococcus, which always proved fatal when untreated. By injecting ethyl-hydro-cuprein-hydrochlorate before the inoculation 90 per cent. of the rodents were saved, and in 50 per cent. death was prevented when the salt was injected after infection. These results place the action of Morgenroth's agent in a totally different category from that of any known chemical antiseptic. When tested in the treatment of pneumococcal infections in man, though the new drug was found disappointing and was proved to produce optic disturbances, it affords us every reasonable hope that by proceeding on Ehrlich's rational lines a substitution product will be discovered capable of giving equal or better results than the startling effects on mice suffering from the same microbic infection.

A grave obstacle to the acceptance of a purely chemical hypothesis of drug action has hitherto existed in what we may term the bogey of catalysis and cell enzymes. Even this, however, has now been removed by the scientific explorer. The most recent researches into the nature of the highly complex chemical structure of the enzymes afford no clue to the plan of their constitution. All we know about them is derived from a study of their effects. They are to be found in every living cell, and the old view which maintained that all the metabolic phenomena of life were due to the vital activity of the cell is now shifted to the action of enzymes. These, even when intra-cellular, have in many instances been separated from the living protoplasm, though in a state of only comparative purity.

Every known enzyme is undoubtedly in the first instance the product of a living cell, but the same may be said of quinine and strychnine, and the part played by the parent enzyme cell may be conceived by the demonstration of Kobert, who procured chemically active trypsin from the body of a spider preserved for a century and a half in the Nuremberg Museum, as Sehrt discovered active glycolitic ferment in the muscles of a mummy. The researches of Croft Hill and Oswald have proved that the action of enzymes is simply chemical. As pointed out by Wells, enzymes not only hasten the splitting up of organic compounds, but they quicken synthesis, though both processes would go on without their presence, only at a much slower rate of progress. He sums up the results of our recent knowledge thus: "All metabolism may be considered as a continuous attempt at establishment of equilibrium by enzymes perpetuated by prevention of attainment of actual equilibrium through destruction of some of the participating substances by oxidation, or other chemical process, or by the removal from the cell, or entrance into it, of materials which over-balance one side of the equation."

Though enzymes do not act quantitatively, they are, nevertheless, practically identical, pharmacologically, with true toxins, *i. e.* with those chemical compounds which are the free secretion of bacilli. If a full dose of a sterile enzyme be introduced into the circulation, a profound toxæmia, terminating in death, occurs. When a smaller amount is injected and repeated in increasing quantities, immunisation results from the formation of antibodies, which impart an anti-enzymatic property to the blood-serum. This serum, when removed from the vessels of the immunised animal, is found to possess the power of neutralising fatal doses of the enzyme injected into another animal, and appears to behave, in every particular, in the same manner as the antitoxin of diphtheria does with

its toxin. Moreover, the anti-enzymatic action of the serum is rigidly specific, combining only with its own enzyme, as pointed out by Eisler.

It appears, therefore, impossible to escape the conclusion that the ferment contains a haptophore and toxophore group, which combine chemically with receptors in the molecule of the antiferment. This would probably apply also to the action of specific hormones, to which Dixon tentatively attributes the physiological activity of the tissues.

Any survey of the potent factors in recent therapeutic advance would be imperfect without a recognition of those purely physical forces whose fascinating influence is manifesting itself on the mind of every explorer in each of the domains of natural science. I refer to those physical laws whose operations are seen in alterations of surface tension, osmosis, diffusion, dissociation, etc.

Our recently acquired knowledge of these forces sometimes proves a veritable *embarras de richesse* to the pharmacologist. Thus one of the latest arguments used for the support of the old physical view of drug action is derived from the study of the *modus operandi* of the hypnotics and narcotics by applying the theory of Hans Meyer and Overton. This postulates that the selective action of a narcotic depends upon its solubility in the brain lipoids, the more powerful members of the group being those least soluble in water and most soluble in fat. Traube's theory also runs on purely physical lines, since he attempts to explain the hypnotic action by assuming a difference of the surface tension, which alters the velocity and direction of the osmosis.

The applications of such theories may be regarded as an illustration of the invisibility of the wood because of the trees. Such theories merely explain the entrance of the drug molecule to the cerebral cell protoplasm without affording us any help regarding the way in which the narcotic effect is produced. To obtain this we must fall back on the chemical theory of Baglioni, which assumes that the temporary paralysis of the cerebral cell is due to the withdrawal of its life-giving oxygen, since the benzene-phenol compounds, in obedience to the law of relationship between physiological activity and chemical constitution, exert their hypnotic action in inverse proportion to the amount of oxygen present in their lateral radicals. That such reduction explanation is almost certain to be true is proved experimentally by the fact that ether, chloroform, chloral and alcohol reduce the oxidising power of living cells. Moreover, the physical theory of Overton and Meyer fails entirely to afford any explanation of the action of the most typical member of the group—alcohol.

June 23rd, 1913.

A LECTURE

ON

ABSENCE OF ABDOMINAL RESPIRATORY MOVEMENT AS AN INDICATION OF PERICARDITIS.*

By W. ESSEX WYNTER, M.D., F.R.C.P.,
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In January, 1911, I read in the Medical Section of the Royal Society of Medicine a short paper on "Absence of Abdominal Respiratory Movement as an Indication of Pericarditis," pointing out that though, "in the majority of thoracic cases affecting the lung and pleura this is one-sided, and the influence on abdominal movement proportionately less marked," in acute pericarditis the cessation of phrenic action is bilateral, and consequently absence of abdominal movement is as obvious as in abdominal disorders, though rigidity and general tenderness are for the most part lacking.

The two cases first cited illustrated this point in a very striking manner, in that the remarkable stillness of the abdomen led to the diagnosis of acute abdominal lesions, the pericarditis being discovered later.

The first seen, in October, 1905, was a young Irishman in the service of a medical friend, who had been seized with acute epigastric pain while waiting at table. Conspicuous absence of abdominal movement led to the supposition that he had a perforated gastric ulcer, and I was asked to take him into hospital for operation. The observation and inference had been made by an independent local practitioner, and were quite disinterested as regards pericarditis, which was not thought of at the time.

Next morning the pain had subsided, he had slept fairly, and was obviously better, clamouring for food, and impatient of restraint. There was no evidence of free gas in the peritoneum or of local irritation. A routine examination revealed nothing. Next day he got up and went out of his own accord, but experienced a recurrence of his pain.

When re-examined on the fourth day, pericardial

* Delivered at the Polyclinic.

friction was quite obvious. There was then dyspnœa and præcordial pain, the abdomen remaining immobile. Pericardial friction was audible four days. Temperature, 101·4° F.; pulse 96. On getting up a week later temperature rose to 103° and pulse 104, but no further development occurred, and he was able to leave the hospital after a stay of three weeks.

The second case, seen in November, 1909, was a man, æt. 40 years, admitted with complaint of pain in the abdomen extending to the thighs, headache, anorexia, vomiting and parched mouth of two days' standing. Temperature, 102·6°F.; pulse, 92. He lay on his back with the legs drawn up, the right rectus also was tense, and there was some tenderness in the right iliac region. Abdominal respiratory movement was scarcely discernible. The only thoracic sign present was a reduplicated second sound. No local indication of pericarditis then existed. Five weeks after the original attack an operation for appendicectomy was performed, but careful examination revealed no morbid condition, and there were no adhesions. The incision healed rapidly, but after five days there was a rise of temperature, with recurrence of symptoms, and pericardial friction was recognised. The diaphragm was not contracting at all, the abdomen actually sinking in with each inspiration. Pulse 90–100. The stomach was dilated, and its resonance could be traced as high as the fourth intercostal space in the axilla. Breath-sounds were absent at both bases, but there was no complete dulness on percussion. Under the X-ray screen a large shadow could be seen in the heart region, suggesting pericardial effusion. The diaphragm was motionless in the position of expiration. Ten days later movement of the diaphragm was returning, though friction could still be heard over the heart. Breathing became audible at both bases, and the patient was discharged a month after the first appearance of the pericardial friction.

Both these cases illustrate the independent observation of loss of abdominal movement as a precursor of pericarditis, and likewise the temporary remission of symptoms attending rest in bed. They also suggest that inflammation of the pericardium possibly commencing on the diaphragmatic surface caused reflex inhibition of movement some days before the appearance of the ordinary clinical signs over the præcordia.

Ten other cases were subjoined, in which loss of abdominal movement had been conspicuous, some with screen examinations, demonstrating the stasis of the diaphragm, and three showing evidence of pericardial effusion.

In one case the contents of the upper abdomen, such as the kidneys and opaque masses in the intestine, could be seen to participate in the upward inspiratory movement, for in this the diaphragm actually rose with inspiration instead of descending, following the aspiration of the upper chest.

Associated phenomena are dilatation of the hollow abdominal viscera, especially of the stomach, and collapse, with loss of function of the lower lobes of the lungs, with slight upward displacement of the heart.

Loss of abdominal movement, as a sign of pericarditis, is the more valuable in that it may precede and outlast the other indications, and so afford grounds for anticipatory or retrospective diagnosis. The condition of diaphragm paresis affords a reasonable explanation of the cyanosis and dyspnœa so usual in the complaint, and of the upright position so commonly assumed by the patient, the former expressing the effect of respiration restricted to the function of the upper lobes of the lungs, and the latter pointing to an unconscious effort on the part of the patient to utilise the weight of the abdominal viscera to depress the passive diaphragm and enlarge the thoracic area.

Restriction of abdominal movement was apparently more marked with a fibrinous exudate than with effusion, in this respect being parallel to similar affections of the pleura or peritoneum.

The detection of a limited amount of effusion in some of the cases by X-ray examination was a new discovery, though it can readily be understood that two or three ounces of serum, gravitating to the back or bottom of the pericardial sac, would give no evidence of its existence by the ordinary method of percussion over the præcordia.

The conclusion in regard to these cases was that where loss of abdominal respiratory movement was well marked, in the absence of more obvious thoracic and abdominal lesions, pericarditis should be closely looked for, and where this sign was wanting dubious indications of pericarditis might be discounted.

Since formulating this statement, rather more than two years ago, I have met with thirty cases

in my hospital wards, containing fifty beds, in which the loss of abdominal respiratory movement has led to the detection of pericarditis—a number so much in excess of those usually noted, that it at once demonstrates the value of such a sign in assisting to recognise a very obscure condition, and strongly suggests that numbers of instances of pericarditis have hitherto been overlooked. This is not altogether incredible in view of the favourable course usually followed by the commonest form, rheumatic pericarditis, without excess of effusion, and is entirely in accord with the dictum of pathologists, that affections of the pericardium of all kinds are recognised far more frequently in the post-mortem room than in the wards, which is tantamount to saying that even the most severe cases are commonly overlooked during life.

But it is not merely in redressing the discrepancy between the clinical and pathological aspects of pericarditis that this sign is of value.

There are many instances of primary illness otherwise unexplained, as well as exacerbations in the course of other complaints, and terminal phases proving fatal in chronic maladies where the timely recognition of pericarditis, which is the dominant factor, would dispel a great deal of mystery and uncertainty, and might relieve much suffering and even prolong life.

Terminal cases are represented by the only ones, four in number, that proved fatal. None were rheumatic.

A young woman of twenty-one was admitted with puerperal sepsis which proved fatal on the third day after admission.

Abdominal movement was absent.

There was evidence of effusion in the right pleura, and friction could be heard over the pulmonary cartilage. Pericarditis was diagnosed.

At the autopsy the pericarditis was demonstrated. There were 8 oz. of fluid, and one pint in the right pleura containing streptococci.

On May 1st, a young woman, æt. 18 years, was admitted in a state of orthopnœa. The temperature was subnormal; the urine of sp. gr. 1015, free from albumen (30 oz.). There was much restricted abdominal movement, and friction could be heard to right and left of sternum, both pericarditis and left pleurisy being present. She died May 4th, and at a post-mortem examination the lesions of the pericardium and pleura were verified. They were secondary to small white kidney and uræmia, the heart weighing 14 oz.

Two other cases proved fatal in children of five. One complicated with purpura and parotitis, but presenting well-marked friction and cantering rhythm over the præcordia. This was septic. The other displayed not only to-and-fro friction over the heart, but effusion was recognised by the aid of the X-ray screen. Unfortunately she developed acute œdema of the lungs, and died very rapidly. Both cases exhibited marked restriction of movement in the abdomen.

As no post-mortem examination was permitted there was no ocular demonstration of the pericarditis, though no reasonable doubt existed.

In four cases the pericarditis was associated with chorea in children. Two were admitted with this complaint, and evidence of pericarditis, either in to-and-fro friction or cantering rhythm, with loss of abdominal movement.

Another admitted with rheumatism and mitral regurgitation developed chorea, and at the same time loss of abdominal movement was noted, and pericarditis recognised by well-marked friction at the base of the heart. When re-admitted for recurrence of chorea three months later, the pericardium was found to be adherent.

The fourth case admitted with chorea later developed pericarditis and effusion.

Abdominal respiration was abolished, and the X-ray screen demonstrated stasis of the diaphragm and a non-pulsating shadow surrounding the heart, representing effusion. All these patients were discharged convalescent.

Next to post-mortem evidence of pericarditis comes that of paracentesis of the pericardium, very infrequently called for, and still more infrequently performed.

H. M—, a boy, æt. 13 years, was admitted for rheumatism and mitral regurgitation on November 1st, 1911.

There was restriction of abdominal movement and præcordial pain. Temperature, 100° F.; pulse, 108. Friction was discovered at the base of the heart.

On November 3rd an extensive non-pulsating shadow was seen by the X-ray screen in the cardiac area. In spite of local and general treatment by salicylates the area of dulness over the peri-

cardium increased and the patient became much distressed.

On November 13th paracentesis was performed. Under a general anæsthetic, given with great caution, the aspirating needle, four inches long, was introduced half an inch outside the sternum in the fourth right interchondral space, being directed horizontally backwards and inwards so as to graze the back of the sternum. By this path, as you may see by looking at this specimen from the front of the chest, the needle passes internal to the mammary vessels, and, entering the pericardium about the middle line, avoids either pleura.

The needle was inserted about two inches, and without any difficulty; some 2½ oz. of blood-stained fluid were withdrawn.

Immediate relief followed, and the boy was subsequently discharged convalescent.

This and the remaining cases were all rheumatic. These run usually a favourable course, and in most instances being included in the general treatment by salicylates would not greatly suffer if the pericarditis were overlooked, as commonly *happens*.

In a fair number of cases, however, the pericarditis develops late in the attack not infrequently while the patient is regarded as convalescent, and sometimes is the cause of delayed recovery or apparent relapse.

For instance, a man, æt. 27 years, was admitted on December 28th, 1912, having been laid up with rheumatism six weeks. Getting alarmingly worse he was brought to hospital, and it was at once noted that abdominal respiration was absent. There was an apex systolic and a double aortic murmur, which made the detection of pericardial friction increasingly difficult. The screen, however, confirmed the diagnosis of diaphragm stasis and excluded effusion in the pericardium; his recovery took six weeks.

One patient, a young woman, æt. 23 years, was reclaimed from a surgical ward, where she was admitted on account of loss of movement and rigidity in the upper abdomen, suggesting peritonitis.

No physical signs were detected in the chest, but next day there was a well-marked pericardial rub in the second left interspace. This was at first to-and-fro, but persisted as a crackling systolic bruit. I had the confirmation of Sir James Kingston Fowler that this was produced over the auricular appendix.

In six cases pericarditis developed some days or weeks after the patient had been admitted with rheumatism, the routine examination on admission satisfactorily excluding this condition, which, moreover, supervened with rise of temperature, sometimes to 104° F., and increasing rapidity of pulse.

In two cases pericarditis was diagnosed and local remedies applied on the strength of these signs, with loss of abdominal movement, and in both friction developed in twenty-four to forty-eight hours.

One case of double basic pneumonia with left-sided empyema developed pericarditis, indicated by loss of abdominal movement and præcordial friction, which subsided on draining of pleura, showing that, as in the pleura, an effusion of pneumococcic origin need not necessarily be purulent.

Perhaps the most signal instance of the value of this sign occurred in a young domestic servant who has only just left the hospital.

She had been laid up for three weeks with fever, dyspnœa, and dulness at both bases posteriorly, and was considered to have had double basic pneumonia. On the fifteenth day, as the temperature had not fallen and she was still very ill, it was thought she must either have an obscure, possibly interlobar, empyema or acute pneumonic phthisis. There was, however, loss of abdominal movement, and in spite of the long and severe illness she was able to sit up to be examined. Moreover, there had been frequent sweating, the temperature had only once reached 103° F., and she was not as prostrated as would be expected after double pneumonia.

An immediate screen examination disclosed stasis of the diaphragm, a pericardial rub having previously been detected. Local remedies were applied and she made a rapid recovery.

I have little doubt that this was an instance of pericarditis, probably rheumatic, associated with stasis of the diaphragm and collapse, not pneumonia, of the lower lobes.

This altered aspect of the case, based on the abdominal symptoms, made all the difference in the prognosis as well as the treatment of the case, with the happiest result.

I have picked out these cases to illustrate special points. The bulk of those included in the survey of thirty in two years were ordinary rheumatic cases in children and young adults, in whom pericarditis occurred as a complication, and was first suspected on account of loss of abdominal movement, or in whom this sign was noted after finding the pericarditis. But I think they help to prove how constant this sign is, and how very much commoner pericarditis is than was formerly believed.

In conclusion a few words may be added about the treatment of the common form of rheumatic pericarditis. Of course salicylates are the potent remedies, but I have long been convinced, by the frequency with which pericarditis develops in patients already taking salicylates in the course of rheumatism, that the internal administration is not sufficient. In all cases I apply an ointment of ʒij salicylate of methyl in ʒj lanoline to the præcordia, renewing it daily for four or five days, till the temperature and other signs subside. It may be even necessary to omit the internal administration of salicylates if symptoms of over-dosage have appeared, but sufficient is absorbed through the skin to keep up the general effect besides concentrating its maximum influence locally. This remedy is effective in effusion as well as in cases exhibiting only friction, though the screen examinations show that in these fluid in small quantity is often present when quite *unsuspected.*

June 23rd, 1913.

Total Deafness following a Dose of Quinine.—M. J. Ballin reports the case of a girl of 16 who in the course of 15 minutes took 30 gr. of quinine. Two hours later she complained of intense headache and severe tinnitus. There was no disturbance in co-ordination, but she noticed a marked diminution in hearing, which steadily grew worse, so that perception for all sounds was completely lost next morning. She consulted different aurists, and finally Prof. Politzer of Vienna, who pronounced her case hopeless.—*Med. Record*, vol. lxxxiii, p. 1043.

PUERPERAL INSANITY.*

By W. H. STODDART, M.D.Lond., F.R.C.P.,
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GENTLEMEN,—I fear you will accuse me of having attracted you by misrepresentation, because the burden of my theme will be that there is no such thing as puerperal insanity. I mean that there is no disease which one can call puerperal insanity, characterised by a uniform series of symptoms or by any peculiarity affecting the whole of the symptoms, by which we can recognise that the type of insanity is a sequel to the puerperium. If one sees a case of insanity without having any history one cannot diagnose that it is of puerperal origin, there being, as I say, no peculiarity, no symptom, by which one can do so.

It is true, however, that insanity specially tends to follow the puerperium in people predisposed to mental disorder. From a nursing point of view it is well to recognise this fact, because every qualified mental nurse knows there are certain things that have to be investigated when a patient comes under her care with a history that the insanity is a sequel to child-birth. Particularly, the nurse examines the breasts and reports whether they are specially full or hard, knowing that an excessive quantity of milk is not only likely to cause pain, but to give rise to abscess. The other thing that a nurse looks for, apart from a distended bladder, cystitis, etc., is vaginal discharge.

This brings me to one of the common forms of puerperal insanity, viz. the intoxication or infection psychosis. As you are well aware, unless special care is exercised during delivery the patient is liable to septic infection. This, in certain patients, is likely to give rise to a form of insanity, which one usually calls "acute confusional insanity." It may also arise from various other infections and intoxications. But the special intoxication which in puerperal states gives rise to it is sepsis.

Such patients are more or less delirious. If it is reported that a patient has a discharge, of course she will be treated accordingly with antiseptic douches, and perhaps an antistreptococcic serum,

* A lecture at the Polyclinic.

But in certain cases the sepsis has given rise to insanity, and no discharge can be discovered. If, however, you are told that a patient has just been confined, and she is suffering from acute confusional insanity, you can often form a very good opinion that sepsis is the cause of it. By confusional insanity, of course, I mean mental disorder characterised by confusion of ideas, the patient having no idea where she is, and perhaps not knowing her own name. In many cases she forgets she is Mrs. "Somebody" and goes back to her maiden name. She suffers from loss of memory and from hallucinations of all the senses, especially of hearing and vision. She is unable to tell you anything that has happened. There is also incoherence merely from confusion of ideas; she is incapable of carrying on an intelligent conversation. She is "wet and dirty" in her habits, not only having no volitional control over evacuation of the rectum and bladder, but even playing with her fæces without realising that such is a filthy habit. In many cases she is destructive. The patient is completely lost, so to speak, and has practically no clear mentation whatever.

If this form of insanity, as I say, arises from sepsis, in my experience the outlook is very unfavourable and the patient seldom recovers. If sepsis has given rise to the insanity, dementia supervenes in the course of some years and the patient usually becomes a permanent inmate of some institution for the insane.

The same series of symptoms may arise, however, not from intoxication, but from mental shock of various kinds; and from examination of the symptoms one is unable to say whether the cause is mental shock or sepsis. It is quite true that Bonhoeffer has written a book in which he tries to draw a distinction between various forms of acute confusional insanity arising from various causes—measles, scarlet fever, influenza, sepsis, mental shock, physical shock, hæmorrhage, and so forth; but from his description I can see no real difference and, in my experience, it is impossible to tell what has been the cause from an examination of the symptoms. All these cases show the same symptoms.

I would impress upon you the importance of another symptom—in addition to those I have already mentioned—namely, analgesia. It is very notable that in these cases a pin-prick is not felt in the arms and legs. In some patients the analgesia is more extensive.

The form of puerperal insanity arising from sepsis is more common than the form arising from shock. Acute confusional insanity arising from shock in puerperal cases is rather uncommon. It occurs in many cases of illegitimacy, there being a very severe mental shock which may give rise to an attack of acute confusional insanity; but the prognosis is that the patient will recover under suitable treatment. The treatment consists of rest in bed, usually with hand and spoon-feeding and, in many cases, tube-feeding for several months together before the patient is able to feed herself. So much for acute confusional insanity.

In patients who have a history of insanity in their family, there is another series of disorders likely to arise, viz. the constitutional psychoses. These are: maniacal-depressive insanity and dementia præcox especially, and some others occurring less commonly as a sequel to childbirth. You will generally find that in about 70 per cent. of these cases there is a history of insanity in the family. In other insanities heredity is much less frequent. It is about 40 per cent., or less, in acute confusional cases.

You may find that the patient, when she comes under your observation, is suffering from mania or melancholia. Mania is a condition of excitement characterised by excessive movement at the large joints. The patient waves her arms and struts about the grounds or ward with unnecessary movement at the hips, swaying the body from the hips and so forth. Mentation is perfectly clear and there is no loss of sensation. The patient knows where she is, her memory is clear, she does not suffer from hallucinations, her perception is clear and she is capable of realising the nature of her surroundings. In many cases perception is even excessive. Such patients make discoveries in looking round their room or ward; they observe things in their room that they never noticed before when their perception was not so brilliant. The medical officer going his round in the morning will hear many home truths, not always agreeable, from these maniacal patients. They are keen in their perception and have not sufficient control to keep their observations to themselves. They are excited, voluble and talkative; they are always busy, especially in the ward; busy chatting pleasantly to

other patients, often helpful and agreeable to the nurses. They are a little mischievous at times, putting things on the fire, turning out the electric light when they know it will cause a certain amount of inconvenience, and generally turning things topsy-turvy, just for fun. But they know what they are doing, and if they think that harm may result from their little practical joke, they will tell the nurse straight away what they have done.

Occasionally one obtains a history in such cases that the patient has been rather violent with her child, wanting to hurl it out of the window, to dash it against the wall, or to get rid of it in some way or other. Happily, this is uncommon.

The patient may be afflicted with melancholia—a condition of depression the exact converse of mania—in which the arms are fixed to the side and there is general paralysis of movement. The patient does not wave her arms about, she feels no inclination to occupy herself in any way, she takes no interest in her surroundings, and suffers from paralysis of emotion and of interest generally. She does not converse with others, and feels depressed. It may sound a little incongruous to say that a patient suffers from paralysis of emotion and yet is depressed. What I mean is that, although the patient is persistently depressed, there is paralysis of emotional reaction. If, for instance, you had to tell her that her husband was dead, she would say, "That doesn't seem to bother me now"; or if you were to tell her a joke that anybody else would laugh at, she would not laugh. This paralysis of emotion sometimes gives rise to certain feelings with regard to the child. The patient suffers from paralysis of emotion, takes no interest in the child and therefore does not want it. Sometimes this lack of interest becomes a positive hatred and the patient is liable to harm the child.

Moreover, a melancholiac is always a potential suicide. There are always social problems in connection with suicide and homicide, and one wonders in such cases whether suicide and homicide are attempts by Nature to get rid of the unfit. The woman suffering from insanity attempts to get rid of herself and also of her child who, after all, is a possible degenerate, having inherited insanity from the mother, or at any rate there is the possibility of its having done so. A point worth noting is that a mother suffering from melancholia of this kind may go round and kill all her own children, but never touch her stepchildren—who are not blood-relations to her. However, this is a digression.

With regard to maniacal-depressive insanity, patients suffering from it are liable to develop mania or melancholia. Such a patient will develop, after the birth of each of her children, an attack of mania or melancholia. It may be always mania or always melancholia, or sometimes mania and sometimes melancholia. But maniacal-depressive insanity has nothing specially to do with childbirth, except that the puerperium is an accidental exciting cause of a particular attack. In many cases the patient has already had attacks of mania or melancholia before you are called to attend the symptoms of the melancholia or mania following on childbirth. Nevertheless, there is undoubtedly a great metabolic change in a woman at the time of the puerperium, and hence we must regard that as a definite exciting cause of the attack. You are therefore justified in giving advice that a woman who is subject to these attacks should have no more children. In some cases one has prevented the recurrence of an attack of insanity by getting the mother to adopt that advice. I am talking of women who have gone twelve or thirteen years without having had another child, nor have they had another attack of mania or melancholia.

There is a variety of maniacal-depressive insanity, which is called periodic insanity, in which the patient has an attack of mania or melancholia at regular intervals, or of mania alternating with melancholia at regular intervals. It does not matter what you do; an attack will most certainly make its appearance at that stated interval. Let us say, for example, that you have a history of a patient having had an attack of mania every two years. You therefore know that every two years the patient will have another attack of mania, and it may occur at childbirth or perhaps a little earlier than childbirth. With regard to these attacks of mania or melancholia in periodic insanity, I am rather a fatalist. It looks as though the person must have the attack, and nothing you can do seems to be able to avert it; the attack most certainly comes on. The interval may be three weeks or any longer period. I have known patients have an attack of mania every three weeks right through life up to sixty years of age. The previous history, before they came under my own observation, of course,

was that they had gone on for forty years, having an attack of mania every three weeks during the whole of that time. The interval may be of any length, from three weeks up to eleven years. Quite recently I have had two patients, each of whom has suffered from an attack of melancholia every eleven years. One of these cases is a lady who had an attack at nineteen years of age, then at thirty, then at forty one, and again at fifty-two—every eleven years—and I expect she will have another attack when she is sixty-three, if she lives long enough. By that time you may say that suggestion has done it. The other case is a person who has just come under my observation again, and who had an attack in 1880, another in 1891, a third in 1902, and a fourth this year. Mania and melancholia may recur in this way quite independently of puerperal insanity.

Dementia præcox may supervene on a puerperium. The patient is perfectly well up to the time of childbirth, and afterwards becomes seclusive, shy and sullen, and takes no interest in people surrounding her or in the child or in her husband. She develops hallucinations of hearing and the disease progresses in the usual way, the patient developing mannerisms and peculiar antics. She gradually develops delusions of persecution, and these delusions of persecution are subsequently replaced by delusions of exaltation. Such is a characteristic history of dementia præcox; but though you examine most carefully a patient suffering from post-puerperal dementia præcox, you will find nothing to prove to you that the disease resulted from childbirth. This class of patient becomes demented and has to be placed permanently under care.

You also come across cases of psychasthenia and neurasthenia following puerperium, but not very commonly.

I have likewise seen general paralysis come on after childbirth. The patient has apparently been perfectly well up to that moment and then begins to develop symptoms of forgetfulness, general loss of memory, perhaps hallucinations of hearing, and excitement, as a rule, in these cases, but possibly depression. On examining the patient you find that her pupils do not react to light; the symptoms also include loss of knee-jerk, tremor of the fingers, tremor of the tongue, imperfect speech, and in fact all the characteristic symptoms of general paralysis. If you examine the cerebro-spinal fluid you will find lymphocytosis, excess of globulin, and a positive Wassermann reaction. General paralysis, then, may supervene on the puerperal state, just as the other insanities may.

There is a condition known as epileptic insanity, and it would appear that epileptic insanity may begin with the puerperium. One of the most remarkable cases I ever came across was a patient who had her first fit certainly within an hour after the delivery of the child. Subsequently she had regular epileptic fits, and in the course of some three or four years became insane, suffering from epileptic insanity. In most cases the epilepsy begins some considerable time after the puerperium. One might almost say that such cases were what used to be called, in olden times, lactational insanity. The patient becomes exhausted from lactation, convulsions occur and epileptic insanity shortly supervenes, leading ultimately to hopeless dementia.

The question arises whether an alcoholic insanity can develop after childbirth? One knows that among the lower classes the tendency to alcoholism comes out especially during pregnancy. People take alcohol, not because they feel any special desire for it, but because they think it is good for them to take a larger amount of alcohol than usual to prepare themselves for childbirth. One would imagine that after a prolonged bout of alcohol such as this, such a traumatism as childbirth would give rise to delirium tremens. We know that, in a chronic alcoholic, traumatism of any kind—a broken leg, or a fall—may start an attack of delirium tremens. But I have never heard of a case of delirium tremens following childbirth. The hæmorrhage resulting from the traumatism in these cases gets rid of the poison presumably in the interests of the child.

Is there any special treatment that should be adopted in a case of puerperal insanity? I am using the term, although, as I said at the beginning, there is no such disease. In the septic cases you can adopt a specific means for overcoming the streptococcus or staphylococcus, as the case may be, by taking a culture and producing a serum and treating the patient with it. But in the cases in which I have done this I have not saved the patient's mentation in any case, though I have saved her life. The outlook in these cases is, therefore, not favourable. You must, of course, look after the physical condition, just as you would in any puerperal woman. You must attend to the breasts by getting rid of the milk by suction apparatus, belladonna plasters or some other means. Then you would naturally treat any discharge and keep the vagina aseptic. Otherwise one cannot recommend any special treatment for these puerperal cases. Rest in bed, of course, is imperative, and proper feeding. Look after their sleep. If they have any difficulty in getting to sleep, give paraldehyde; if they wake too early, give amylene hydrate, sulphonal, veronal or chloral, according to the particular requirements of the case.

As I said at the beginning of these few remarks, I have appeared before you under somewhat false pretences. I undertook to lecture on "puerperal insanity," but the burden of my theme has been that there is no such thing.

June 23rd, 1913.

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CASES ILLUSTRATING CERTAIN URINARY CONDITIONS IN WOMEN ASSOCIATED WITH FREQUENT OR PAINFUL MICTURITION.*

By DAVID NEWMAN, M.D., F.R.F.P.S.G.,

Surgeon, Glasgow Royal Infirmary.

A CONSIDERABLE number of women suffering from frequent or painful micturition fail to gain any permanent improvement even after most careful treatment, and not uncommonly the patient is sent with the report from her medical adviser—"I suspect a stone in the bladder, but have not been able to find one; I would like a cystoscopic examination made, and an opinion as to the cause of the trouble."

Undue frequency of micturition accompanied by pain, or what was formerly known as "irritability of the bladder," is the first indication of many urinary diseases, not of the bladder only, but also of the kidney. That a routine and complete examination is necessary to the proper understanding of what may appear at first sight to be a simple case is forced upon the surgeon the more he studies the details of urology. In the great majority the diagnosis is easy and moderately certain, and one is apt to be satisfied when a lesion is discovered which may explain the systoms. The treatment is, however, not successful; something has been left unseen. As with the interior of the eye, the ear, or the throat, the lesion in the bladder must be seen. Who would give an opinion of a diseased optic disc without using an ophthalmoscope? But still men express freely their opinion of a diseased bladder without looking at it.

* A communication made to the Medico-Chirurgical Society of Glasgow.

Symptoms, no doubt, often indicate clearly the nature and site of the lesion, but just as often in the urinary tract they mislead, unless resort is had to physical proofs to guide and correct the conclusions deduced from the subjective phenomena. There are some diseases more liable than others to lead us astray. To only six of these will our attention be now directed, namely:

(A) Cystitis of pregnancy, with its results.

(B) Early renal tuberculosis.

(C) Early tuberculosis at the base of the bladder.

(D) Lesions at the neck of the bladder, the trigone, and the urethra. Incontinence of urine.

(E) Polypus of urethra.

(F) Movable kidney.

The study of our own mistakes and the mistakes of others demonstrate that these lesions are not kept clearly enough in view in making a diagnosis. It is essential to recognise that such conditions exist, and are met with frequently in practice. Where the subjective symptoms are accompanied by well-marked objective signs, the diagnosis is easy and at once apparent, but it is otherwise when there is no distinct change in the character of the urine, and no physical signs pointing to the seat of the lesion.

(A) *Cystitis of pregnancy, with its results.*—This form of inflammation of the bladder is of interest as the forerunner of trouble in a great number of women, for the reason that the symptoms during pregnancy often are not very severe, and are attributed by the patient to "her condition." Herein lies the danger; often she says little or nothing about it to her medical attendant, and it is only when questioned, perhaps years after the infection of the bladder has been established, that she recalls the onset of the disease. When treated at the beginning the cure is easy and no harm results, but if left alone much worry and pain is the reward of neglect.

Following chronic constipation and engorgement of the colon in young women about the fifth or sixth month, a moderately acute attack of cystitis sets in. The onset is sudden—an acute rigor, discomfort over the bladder and hypogastrium, painful and frequent micturition, temperature elevated and irregular, pulse rapid, and tongue furred. Soon the patient complains of wide-spread pain in the back, loss of appetite, headache and depression, and all the symptoms of toxæmia. On examination, the patient complains of pain on pressure over the kidneys and bladder; the kidneys are found to be enlarged, and there is tenderness and rigidity in the lumbar muscles; and leucocytosis, polymorphonuclears and red blood-corpuscles show evidence of anæmia. The urine is acid, smoky, contains some albumen, pus, and squamous epithelium. The odour is not offensive at first, but on standing soon becomes so. Such a case demands attention, and probably gets help. It is in cases where the attack is not severe that danger lies, or when the acute symptoms disappear in a short time, and care is not taken to free the bladder from infective organisms. Women so attacked during pregnancy are liable to suffer after delivery from continued irritation of the bladder, which is only too liable to be left unheeded. When the disease becomes chronic, gradually the symptoms may become so severe as to give rise to the suspicion of stone, but not finding one on sounding, the family doctor may satisfy himself, but not the patient, with palliative treatment, which is inadequate for the condition. An examination shows the suffering to be due to inflammation of the neck of the bladder or the urethra, lesions which will be considered presently.

CASE 1.—*Bacillus coli infection of bladder at fifth month of pregnancy—Incomplete recovery; continued irritation of bladder during five years, slight pyuria in acid urine; slowly and steadily increasing pain during and after urination—Thickening of mucous membrane at the neck of bladder and urethra—Application of phenol to urethra, and flushing of bladder—Cure.*

Mrs. R. S—, aged 35 years, was admitted to the Woodside Home on September 4th, 1909, and recommended as a case of stone in the bladder. The history showed that for five years the patient had suffered from slowly and steadily increasing micturition, while during the last year she passed urine every hour in the daytime, and as often as six or seven times during the night. Rest in bed and warm applications gave relief, but as soon as she began to move about the pain returned. (This fact gave rise to the suspicion that a stone was present in the bladder.) The urine was highly acid, there was a considerable deposit of uric acid, and small quantity of pus was always present.

On cystoscopic examination no stone could be seen, and no shadow was obtained in the renal region with the X rays. The mucous membrane was slightly injected throughout the whole of the bladder, and at the neck there was very marked congestion and thickening of the tissues (Fig. 1, *a*, *b*, and *c*), which formed three distinct nodules. These nodules were very sensitive, and when a bougie was passed over them great pain was complained of.

Cultures from a catheter specimen of urine gave pure *Bacillus coli* from the bladder, but a mixed infection was found in the urethra.

The mucous membrane of the posterior two thirds of the urethra was deeply injected, but no ulceration could be discovered. No tubercle bacilli and no gonococci.

The bladder was washed with boracic acid solution twice daily, and after a week the neck of the bladder and the urethra were swabbed over with pure phenol, and afterwards with an alkaline solution to stop the action of the acid. Cocaine bougies were introduced to relieve pain. Result satisfactory. Reported cured, October 26th, 1909.

Well and last seen, April 5th, 1911.

Those who are familiar with the varying conditions and reactions of the urine in different healthy individuals, and of the same person at different times, must have observed that some samples undergo decomposition with great rapidity, while others remain unchanged for a considerable time. Early decomposition of urine, which is sterile when excreted, is due to the fluid containing waste products capable of easy decomposition. Consequently, we find that when a woman is suffering from imperfect digestion, nervous disturbance or constipation, the urine decomposes early. During pregnancy the excretions are modified considerably, and the urine is rendered a more suitable medium for the proliferation of organisms. Since the time of the ancients changes in the urine have been noticed, as well as other phenomena having origin in the glandular system, but even now the peculiar liability of the bladder to *Bacillus coli* infection during pregnancy has not been sufficiently emphasised.

For obvious reasons, the female bladder is very liable to infection along the lumen of the urethra. Hence infection is generally ascending, whereas in man it is descending in character. In women

FIG. 1.—*Bacillus coli* infection of bladder, showing three highly sensitive nodules at the neck. The mucous membrane was deeply congested and thickened.

urethritis and cystitis are often met with, independent of any renal lesion, whereas in men they are most frequently associated with some septic disease of the kidney.

Unfortunately, a general belief prevails that urethral inflammation in women is necessarily gonorrhœal in origin ; but, while generally it may be so, a considerable number of very troublesome cases are met with in which gonococcus is not the infective organism. The most common of these is that following the cystitis of pregnancy, and it is nearly always a *Bacillus coli* infection. When the onset of the disease is acute and well-defined the diagnosis is made at once, but if the infection be mild and the development of the lesion slow, things are allowed to drift until the symptoms become severe, and by that time the relation of the cystitis to pregnancy is liable to be forgotten.

The cystoscopic appearances do not, as a rule, show any serious changes in the mucous membrane of the bladder ; indeed, they are often surprisingly slight considering the amount of pain and inconvenience they occasion. It is the situation of the lesions, not their severity or extent, that causes the trouble. There may be simply patchy congestion without much thickening of the mucous membrane, and in afew instances the lesion ma y escape the notice of an inexperienced observer (Fig. 2). For this reason the surgeon must be most careful in his cystoscopic examination, especially of the base and neck of the bladder. In the great majority of cases, however, the evidence of disease is more obvious, the mucous membrane being congested and covered with thin flakes of muco-purulent

FIG. 2.—*Bacillus coli* infection, showing hyperæmia at the neck of the bladder, but so slight and limited as to be liable to escape observation.

FIG. 3.—*Bacillus coli* infection. The mucous membrane deeply congested and covered by flakes of muco-purulent material, which are usually adherent to the œdematous and swollen mucous membrane.

material (Fig. 3), which, being adherent to the epithelial surface, are not readily dislodged by washing. *Bacillus coli* infection of the bladder, when allowed to persist, is very liable to induce urethritis, which is often very troublesome, but the secondary results will be described under section (D).

(B) *Early renal tuberculosis* is the next condition which calls for attention. In young women frequent or painful micturition is often the first indication of disturbance in the urinary tract, but its importance as a symptom is liable to be overlooked. The following passage from an article by the writer may be recalled : " Indeed, when, independent of any vesical or urethral disease, frequent micturition or nocturnal incontinence is complained of in young persons, the symptoms should always arouse suspicion, and, even although the patient does not complain of pain in the bladder, a careful inquiry should at once be made to demonstrate the cause of the symptoms." The early tuberculous lesion in the kidney induces increased functional activity of the affected organ, as shown by polyuria, and reflex irritability induces the bladder more readily to respond to moderate distension. On the other hand, the frequent micturition in tuberculosis may be a consequence of secondary infection of the bladder, which is often an early accompaniment of the renal lesion. And here also the seriousness of the symptom is apt to be underrated from the circumstances that the micturition is not accompanied by pain. The patient does not regard it of importance, and fails to mention it to her medical adviser until other symptoms develop. Vesical irritability, and after a time pain also, become more or less prominent features ; often the irritability is limited to the early hours of the day, and becomes less as the day advances. When the bladder is distended to a certain degree the pain may suddenly come on, demand immediate emptying of the bladder, and may continue for some time after micturition is completed. As the disease advances generally a slow process at first—the pain, the frequent urination and vesical tenesmus steadily become worse. The character of the pain is variously described as sharp, aching, dull, and may be transmitted along the urethra to the renal region, or even over the whole of the back. But usually it is limited to the bladder and suprapubic areas. The amount of pain depends more upon the locality of the lesion in the bladder than its extent—when the upper and middle zones alone are implicated the suffering is slight, whereas if the disease involves the base, and especially the neck, pain is a very prominent symptom.

The premonitory symptoms of early renal tuberculosis and primary vesical tuberculosis are very similar—in fact, all the symptoms of the latter may be present when the kidneys alone are involved, and, on the other hand, considerable disease of the bladder may exist without giving rise to any trouble. This is, however, only met with when the neck of the bladder is not included in the lesion. Remission of the symptoms is one of the most characteristic features of the disease, both as it affects the kidneys and the bladder. During a remission the patient's general health may improve, she may gain weight, her temperature remain undisturbed,

the polyuria, hæmaturia, and frequency of micturition may disappear, and she may suffer no pain and little inconvenience. This favourable condition does not last long, and sooner or later all the old troubles reappear with fresh vigour. When a remission occurs, especially if it be a long one, a hopeful medical attendant may be too easily led astray by the belief that things are improving, and that the symptoms after all may have been due to a trivial cause. In all cases an exhaustive examination is demanded, and the familiar objective methods should be employed:

(1) Bacteriological examination of the urine and inoculation experiments.

(2) Ophthalmo-tuberculin reaction.

(3) Segregation of the urine.

(4) Cystoscopy.

(1) The discovery of tubercle bacillus in the urine is of first importance, but as they may be present in small numbers, repeated examinations may be made before a reliable opinion can be formed simply by microscopic examination. In such cases inoculation experiments clear up the diagnosis, as when tuberculous urine is injected into the peritoneum of a guinea-pig a tuberculous peritonitis is sure to follow within a month or six weeks, or earlier.

(2) Ophthalmo-tuberculin reaction is also a useful, although not an absolutely reliable test. Usually in tuberculous subjects in eighteen to thirty hours after the installation distinct hyperæmia appears at the caruncle, and in forty-eight hours conjunctivitis results. It only proves that the patient is tuberculous; it does not locate the lesion.

(3) Segregation of urine is not to be recommended in early tuberculosis unless in exceptional cases. Undoubtedly catheterisation and segregation are of value in order to obtain reliable information as to the kidney which is diseased, as well as an estimate of the functional activity of its neighbour; but unless ordinary clinical methods, aided by the careful use of the cystoscope, fail to supply the information required, these methods should not be resorted to in early tuberculosis. Considering the amount of information that may be obtained from a cystoscopic examination, the cystoscope should always be used before ureter-catheters, or segregators are employed.

(4) Cystoscopy affords most useful and reliable information.

In early tuberculosis of the kidney the following conclusions may be accepted: (*a*) When one ureteral orifice is altered while the other is normal the renal lesion is on the side of the morbid ureter; (*b*) the deformity of the orifice and the lesion there indicate the nature of the renal disease; (*c*) the character of the urine escaping from the ureter denotes the morbid changes taking place in the corresponding kidney; (*d*) the frequency, size, and regularity of the "shoots" from the two orifices indicate the functional activity or the presence of undue irritation in the respective kidneys; (*e*) when the orifice of the ureter is strictly normal no serious disease exists in the corresponding kidney; (*f*) when the kidney is normal the orifice of the ureter is also normal; (*g*) when there is evidence of tuberculosis at the orifice of the ureter there is always associated with it tuberculosis of the corresponding kidney; (*h*) in tuberculosis of the bladder the ureter does not become involved if the corresponding kidney is free from disease.

(c) In *early tuberculosis of the bladder* the most characteristic changes are hyperæmia of the floor and neck of the bladder associated with the deposit of minute nodules beneath the epithelium of mucous membrane of the bladder. These are at first clear, like boiled sago grains, but gradually become opaque from caseous degeneration while the congestion increases, and the appearance is produced of bright red rings enclosing pale, chalky-looking elevated nodules. Often irregular erosions are covered with white flakes of mucus or muco-pus. Two or three ulcers are usually found in the same bladder, and adjacent to the ulcers miliary tubercles are generally seen. These are the appearances in the early stage, when the disease is liable to be overlooked; later on the diagnosis is only too easy. The premonitory sign, frequent micturition, and especially nocturnal micturition in young women, may exist long before any other complaint is made, and is generally associated with increase in the amount of the excretion, but not being accompanied by pain the patient does not regard it as of any great consequence, and her medical adviser is not consulted. It is only when vesical irritation and pain become prominent or hæmaturia occurs that alarm arises in the mind of the patient or her friends. These symptoms generally indicate that

the premonitory stage of the disease has nearly passed, and the period open to successful surgical treatment will soon be over.

CASE 2.—*Frequent urination for two years, with pain only on distension of the bladder, disappearing when emptied—Latterly, pain always present—Moderate pyuria; 5 per cent. pus containing tubercle bacilli; no hæmaturia—Marked hyperæmia of bladder and caseous deposit on floor—Curetted through urethra—Tuberculin R.—Well, 1911.**

Patient, K. S. B—, æt. 24 years, was admitted to the Glasgow Royal Infirmary on November 5th, 1906, complaining of pain, frequency of micturition, and a bearing-down feeling in pelvis of two years' duration.

History of present illness.—About two years ago she first noticed a desire to pass urine oftener than usual, and had much pain whenever there was any distension of the bladder. At first the pain disappeared whenever urine was passed. This frequency of micturition became worse, until last May, when she had to pass urine every hour and a half. Since then frequency has lessened, and she can now retain urine for about three hours, but the pain is always present when she is walking about, and does not disappear, as formerly, as soon as the act of micturition is completed. The pain disappears to a large extent when she goes to bed, but the increased frequency of micturition remains. Through the present illness the patient has not been losing weight, but occasionally has "feverish attacks." Except when she had an attack of pneumonia last summer, she had not noticed any swelling of the feet or eyelids.

Previous health.—Patient had scarlet fever when a child, and pneumonia last summer, but otherwise has enjoyed good health.

Present condition. — Patient is fairly well nourished. Heart and lungs seem in good condition. Pulse 84, small in volume and tension, but regular.

Examination of abdomen reveals nothing abnormal to percussion or palpation. Kidneys not palpable, and no tenderness to be found in that region.

Urine.—Pale in colour; acid reaction; specific gravity, 1020; gives reaction for albumen, but not for blood or sugar; microscopically, pus-cells are numerous.

FIG. 4.—Early tuberculous deposit in primary vesical tuberculosis. The mucous membrane was deeply injected, and in the trigone there were four oval submucous caseous nodules, which were removed by curetting.

November 13th, 1906.—Cystoscopic examination by Dr. Newman. The whole of the mucous membrane of the bladder was deeply injected. On the right side of the trigone there were several submucous caseous deposits, elongated, oval in shape (Fig. 4). The caseous matter shone through an unbroken epithelial cover; no ulceration at any point. Tubercle bacilli present in six out of ten examinations. Dr. R. M. Buchanan's report was: "The guinea-pigs inoculated, when killed, were found tuberculous."

November 14th, 1906.—Patient getting 5 gr. phenacetin and 5 gr. ext. of hyoscyam three times a day. Cystostomy not required; bladder curetted by urethra. Tuberculin R. prescribed once a fortnight for next four months.

March 13th, 1907.—Cystoscopic examination by Dr. Newman. A good view was obtained of the bladder, in which nothing abnormal could be found, with the exception of a little hyperæmia at the neck and two small nodules similar to those described in last report. The mucous membrane was pale in colour, and the vessels were not very clearly defined.

May 1st, 1908.—Cystoscopic examination by Dr. Newman. Examination of the bladder showed slight hyperæmia around the neck, otherwise the mucous membrane was normal in appearance, and the tuberculous nodules, referred to in last report, had disappeared.

* This case was reported in an article on "Primary and Early Secondary Tuberculous Cystitis," 'The Practitioner,' July, 1912.

February 19th, 1909.—Cystoscopic examination by Dr. Newman. The mucous membrane of the bladder was normal in appearance, with the exception of some œdema, and the arterioles were not clearly seen. No nodules or ulceration seen.

June 20th, 1910.—Patient reported well, and able to follow her occupation of an hospital nurse. No tubercle bacilli present.

November 20th, 1911.—The patient was reported to be well.

(D) *Lesions at the neck of the bladder, the trigone, and the urethra.*—In women a form of irritable bladder is common where the patient is distressed by frequent micturition during the day, and during the night there may be occasional incontinence. The only complaint may be frequency, and although it may be most troublesome and inconvenient to the patient, from the nature of the illness she is liable to hide or minimise her suffering rather than seek medical advice.

Another condition worthy of attention is persistent incontinence of urine in women.

CASE 3.—*Five years' persistent frequency in micturition—Relieved only by rest and heat—During last three months symptoms resembling stone in bladder—Constant pyuria — Moderate occasional hæmaturia—Neck of bladder and urethra congested, and small superficial erosions—Phenol applied twice—Cure.*

Miss W—, æt. 58 years, was sent to me with a long history of bladder trouble, persistent frequency, and painful micturition with slight pyuria. On four occasions she noticed some blood in the urine, but her medical attendant informed me that on many occasions he had discovered small quantities of blood. The only treatment which afforded any relief was complete rest in bed, and continued application of hot fomentations.

On admission to the M'Alpin Home a cystoscopic examination showed marked congestion of the neck of the bladder and upper portion of the urethra, with several small superficial erosions in the latter region (Fig. 5). The infection was due to *Bacillus coli*, and no tubercle bacilli or gonococci were found.

The whole course of the urethra and the mucous membrane at the neck of the bladder were on two occasions swabbed with phenol, and afterwards with an alkaline solution. Considerable pain followed the application, but it was relieved with cocaine bougies.

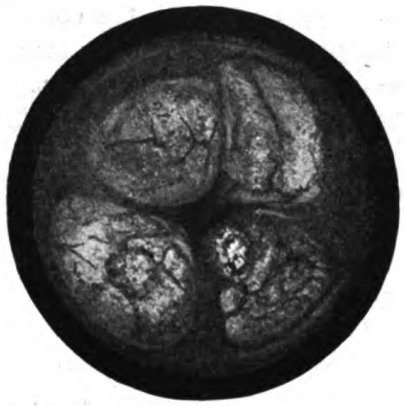

FIG. 5.—*Bacillus coli* infection of urethra. Shows upper portion of urethra, with marked thickening and congestion of mucous membrane, and several small superficial erosions.

When admitted she was passing urine from eighteen to twenty-two times in twenty-four hours. When dismissed six weeks later she could retain urine for three hours during the day and considerably longer during the night, and all pain was gone.

The condition being associated with the excretion of highly acid urine, its true cause is likely to be overlooked, and the reaction of the urine blamed rather than an infection of the vesical triangle or neck of the bladder. The floor and the neck of the bladder become over-sensitive. To understand this disorder, which is so often the cause of involuntary micturition in children and adolescents, it may be well to recall the conditions which control normal micturition. Under higher centres in the cerebrum there are two centres in the lumbar region of the cord, a sphincter centre and a micturition centre—the former controlling the muscular fibres at the neck of the bladder, the latter the muscular structures of the bladder-wall. When distension of the viscus occurs, the mucous membrane becomes stretched and the sensory nerve-fibres are stimulated, and through the centre in the cord an impulse is conveyed to the sensorium. On perception of this, a double impulse is emitted from the brain to the cord—one to stimulate contraction of the muscular fibres of the bladder-wall, the other to inhibit the sphincter centre, and thereby induce relaxation of the neck of the bladder, and so permit of the escape of the contents of the viscus.

In involuntary micturition the conditions are different: the passage of urine takes place without the intervention of the higher centres. This is seen during the early months of life. The infant passes urine every half hour while awake, and during sleep probably every two hours. But gradually as the child gets older the intervals lengthen, and the infant acquires, with proper training, some control over its bladder, so that in a few months, or, if the mother is careful in training, in a few weeks very few napkins need be wetted. In children whose training has not been attended to, where they have not been taught to pass urine at regular intervals, the napkins are seldom dry for any length of time. This is a fruitful source of infection.

A careless mother or a nurse, in place of supplying clean napkins, simply dries the soiled ones, using them several times without washing. These cause scalding of the perinæum and adjacent parts, including the vagina and urethra, followed by infection of the urethra and symptoms of cystitis. When the attack subsides a certain amount of bladder irritation remains, and as the patient passes from infancy to childhood nocturnal incontinence manifests itself, and very commonly the sufferer is blamed for carelessness not her own by those who should not have allowed the infection to have taken place.

Nocturnal incontinence in young girls (not in boys—with them the trouble is due to other causes), which continues in spite of all care and treatment, becomes a source of anxiety and annoyance to the parents, as well as of discomfort and discredit to the child. During deep sleep urine is passed, and the child is conscious of its escape only when she awakes. The flow is not due to inability of the bladder to retain an ordinary quantity of urine, but rather to undue readiness to contract when the reflex action of the vesical muscular coat is uninfluenced by the will.

What seems to happen is that just before waking, when the bladder is distended, reflex action is set up and the contraction of the sphincter is inhibited, and the urine escapes, or the control of the will being in abeyance a very slight distension of the bladder calls forth involuntary micturition, which may be repeated several times during one night.

Cystoscopy shows in many of these cases a congested neck and slight hyperæmia of the mucous membrane, the urine contains an undue quantity of clear mucus, a deposit of epithelium, and *Bacilli coli*. On adopting suitable local treatment, the disagreeable symptoms disappear.

CASE 4.—*Involuntary nocturnal micturition since infancy—Complete control during the day and while awake, no pain, occasional slight pyuria, very acid urine and uric acid in considerable amount—Irrigation and installations—Cure.*

Miss A. P—, æt. 19 years, consulted me on August 26th, 1908, on account of involuntary micturition during sleep which happened several times a week and had gone on as long as she could remember. All kinds of treatment had been employed both at school and while she was at home, but no lasting benefit was obtained. As far as can be made out the bladder empties itself immediately before she awakes, or more probably the wetting awakes her, but it is at her hour to awake, between 7 and 8 o'clock a.m., that the accident nearly always happens. But if she is roused, say at 5 o'clock, before she can get out of bed the bladder has emptied itself. Careful observations were made by waking her during the night and measuring the quantity of urine passed each time, and it was found that she could retain ten ounces, but if the bladder contained more than that amount excavation was certain to take place on awakening from sleep. No diseased condition external to the bladder could be discovered, and the patient could not be called "neurotic" in the least degree. The urine had not been examined bacteriologically, nor had a cystoscopic examination been made. The patient enjoyed perfect health, was well developed, and very fond of active exercise.

Cystoscopic examination showed distinct congestion at the neck of the bladder (as in Fig. 2), and slight hyperæmia of the uppermost third of the urethra. Otherwise the urinary tract was normal. The urine was very acid when passed, and shortly thereafter deposited crystals of uric acid, clear mucus, and a small quantity of pus. Pure cultures of *Bacillus coli* found on all occasions.

The bladder was carefully irrigated with a sterile solution of potass. permanganate (5 gr.) and potass. chlorate (10 gr.) to the ounce of water, at first by fully distending the bladder, afterwards by con-

tinuous irrigation. This treatment was continued for a fortnight, the patient being kept in bed; then, in addition to the irrigation, instillations of nitrate of silver were employed, 2 gr. to the ounce of water. This was repeated six times, and the patient was sent home on September 30th, 1908.

June 19th, 1909.—Only three accidents have happened since September, 1908.

In other cases the patient has complete control both while awake and during sleep, but on account of irritation she requires to rise several times during the night, and in the day is in trouble also. The irritation at the base and neck of the bladder may be complicated by reflex irritation induced by —(1) Cystitis; (2) vulvitis; (3) caruncle; (4) ureteritis; (5) fissure at neck of bladder; (6) highly acid urine; (7) renal disease; (8) endometritis; (9) onanism.

These conditions are not likely to be overlooked if they are kept in mind. There is only one lesion which often escapes notice, simply from the fact that the patient is not submitted to a careful endoscopic examination. *Inflammation of the female urethra* is a condition of much greater frequency than is generally believed, and because the urethra and neck of the bladder are not inspected the case is wrongly diagnosed, and the suffering of the patient attributed to cystitis, stone in the bladder, or renal calculus. Urethritis, while most frequently gonorrhœal in origin, frequently exists in women who have had no opportunity of infection, and where no gonococcus can be found in the discharge or urethral mucus. The inflammation is caused by septic or *Bacillus coli* infection. On examination with the endoscope various conditions may be met with.

(1) The mucous membrane may be uniformly infected and swollen, and the slightest injury of the mucosa will cause enough bleeding to obscure a view.

(2) In other cases the surface is mottled, and the mucous glands stand out as pale bodies on a dark infected background. On pressure a thick muco-purulent discharge may be expressed from the crypts.

(3) In severe and acute lesions the mucous membrane may be covered by a membrane composed of fibrin and epithelial *débris*, which when removed exposes an eroded surface.

CASE 5.—*Long-standing Bacillus coli and staphylococcus infection of bladder—very frequent micturition and severe pain, hæmaturia and pyuria variable —Marked congestion of mucous membrane of the bladder and urethra—Suprapubic cystostomy, and phenol applied to neck of bladder and urethra—Almost complete relief of symptoms.*

Mrs. M'K—, æt. 45 years, consulted me on January 24th, 1910, complaining of symptoms strongly suggestive of stone in the bladder, but her family attendant was unable to discover one either with the sound or by the X-ray plates. The symptoms—pain during and after micturition, frequency, hæmaturia, and pyuria—dated fully back nine years, and during that time the symptoms had steadily increased in severity until they rendered her life a constant misery night and day. She required to pass urine every half hour, always with great pain. Temporary relief was obtained from morphia, rest in bed, and hot applications, but as soon as she resumed active life the pain returned.

Examination of the bladder and urethra showed marked congestion of the mucous membrane at the extreme base of the bladder around the neck (Fig. 6), but most acutely throughout the upper two thirds of the urethra. In the urethra the mucous membrane presented a mottled appearance, the ground being of a chocolate colour, and the elevations, little round nodules, being bright red, but not denuded of epithelium. *Bacillus coli* and staphylococcus in urine.

FIG. 6.—Old-standing mixed infection of bladder, showing appearance of the neck, which is completely surrounded by a papillomatous-looking mass of inflammatous exudation. Mucous membrane thickened and congested.

The bladder was carefully irrigated and the patient kept in bed; the usual constitutional and local treatment, including application of phenol to urethra, was carried out without any marked improvement in the symptoms.

February 21st. — Suprapubic cystostomy performed and temporary drainage employed. Bladder washed out twice daily with permanganate of potash solution; later on lactic bacilli cultures introduced and allowed to remain.

March 23rd.—Relief of symptoms. Patient's general health much improved and passing urine six times in twenty-four hours.

The surface of the trigone is one of the most common sites of inflammation (Fig. 7), and demands radical treatment in some instances. Beneficial results are obtained in many by irrigation of the bladder as above described, but in the more virulent infections more is required. A suprapubic drain must be established to give free exit to the contents of the bladder and allow cleansing. A temporary drain of this kind is by far the most effective treatment of chronic cystitis, and even in the most persistent and intractable cases astonishing results are obtained. The urine becomes healthy and the mucous membrane normal, and in a few weeks the wound may be allowed to close. The drainage employed is by syphoning the urine into a suitable vessel, so as to permit the patient to do without dressings.

This drainage must be supplemented by careful irrigation, and if the urine is alkaline, or contains alkaline phosphates after irrigating, an installation of lactic bacillus cultures should be given. Milk whey containing pure lactic bacillus not only destroys the septic organisms in the bladder, but also dissolves phosphatic crusts adhering to the bladder or the wound. The filtrate of saurine does admirably, and may be used with advantage in all purulent conditions. A suppurating sac or a granulating surface thoroughly washed regularly with it cleans up rapidly; it acts more satisfactorily than any chemical antiseptic, and the lactic acid bacillus, not being a pathogenic organism, does no harm. In some cases quite remarkable results have been obtained.

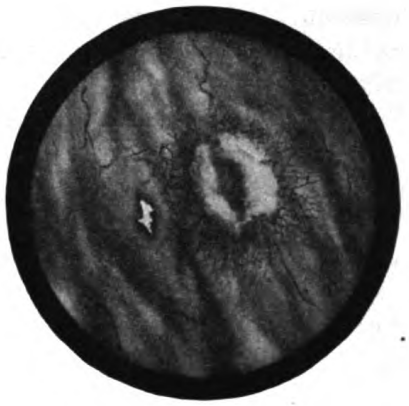

FIG. 7.—Mixed infection, with ulceration of mucous membrane of the trigone. One ulcer is seen clearly, the other, the larger, is completely hidden by a cloud of white mucus.

CASE 6.—*Very painful and frequent micturition from staphylococcus infection of bladder—Pyuria and hæmaturia—Only 2 oz. of urine retained—Acute catarrh of base—Instillation of silver nitrate—Suprapubic drain—Lactic bacillus cultures and irrigation—Cure in six weeks.*

Mrs. M'N—, æt. 40 years, admitted to the Glasgow Royal Infirmary on August 21st, 1912, complaining of very severe pain during and after the act of micturition, so severe as to bring on sickness and severe headache. She had an inclination to pass urine every half hour, but very little came away at each time—not more than 2 oz. The urine was muddy and bad smelling, and was always tinged with blood. The urine when passed was alkaline, contained pus in abundance, and "strings" of blood. The only organism found was *Staphylococcus pyogenes albus.*

August 27th, 1912.—When under an anæsthetic the bladder was able to contain 4 oz. only, but after treatment to September 6th, 7 oz. were retained. An instillation of silver nitrate was used.

October 19th.—Cystoscopic examination. The floor of the bladder is inflamed, and there are a number of small prominences on the surface of the mucous membrane in the trigone. These are coated with phosphates, which are irregularly distributed, forming patches and edges. The pure white phosphatic deposit stands out in marked contrast to the deeply injected mucous membrane.

October 29th.—Suprapubic cystotomy was performed, and a mass of pulpy granulation-tissue was removed from the neck of the bladder.

December 6th. — Bladder washed out with

boracic acid solution and filtrate of saurine (lactic acid cultures) for five weeks, without much improvement in the local pain, but with great improvement in the condition of the urine. Yesterday patient had no pain, and passed urine naturally. Frequency has greatly diminished.

December 10th.—The wound is now closed. Symptoms have undergone sudden improvement. Frequency has been reduced to four or five times a day.

December 17th.—Pain absent for a week. Dismissed.

Non-gonorrhœal infections in women have not attracted the attention they deserve. They are nearly always overlooked, and so give rise to prolonged trouble both to the patient and to the medical attendant.

Incontinence of urine in women. — Married women, and women who have had the urethra and neck of the bladder over-dilated by any cause, are liable to suffer from injury to the muscular structures at the neck of the bladder—the structures which control the discharge of urine.

The escape of urine may be continuous or intermittent, but in all it is very distressing to the patient, and prevents her from associating with her friends. The wearing and perpetual changing of wet absorbent gauze pads prevents the woman from going into society, and the irritating secretion causes inflammation of the external genitals and considerable suffering.

The only remedy for this condition is an operation, the details of which I hope to publish shortly. The object is to reunite the fibres of the sphincter at the neck of the bladder and narrow the urethral canal throughout its course. The operation has been quite successful: patients who have been unable to retain urine in the erect posture, have, after the operation, kept it for two, three, or four hours without difficulty—the control for that time being complete.

(E) Minute polypi in the urethra are sometimes a cause of considerable local irritation and obstruction, but the condition is so rare that it is only worthy of being mentioned, the writer having seen only three instances of this disease. The polypi may occupy the whole course of the passage, but are most commonly seen at the internal meatus, where they appear like small vesicles. When discovered with the endoscope they should be removed with a curette or snare.

CASE 7.—*Sudden stoppage of flow during act of micturition, followed by severe spasm at the neck of bladder—Polypi removed—Cure.*

Mrs. B—, æt. 56 years, complained of difficulty in micturition for many years, but never very troublesome till six months ago, when she consulted me for the first time. The only symptoms were that during the act of micturition the flow suddenly stopped, without pain at first, but within half a minute or so she had an oppressive desire to pass water, but none would come away. By waiting, however, for ten or fifteen minutes usually she succeeded in emptying the bladder completely, but not always, as now and then no urine would come away. The urine was always strictly normal. With the endoscope a large elongated polypus was seen (Fig. 8), and a smaller one. Both were removed with a wire snare, and immediate relief obtained.

Only three other cases may be mentioned.

The first case illustrates a peculiar condition of the lower margin of the neck of the bladder. The lesion consisted of a crowd of very delicate fimbriated processes, with bulbous ends (Fig. 9), extending from the margin inwards for a distance of about a third of an inch; the papillæ were pale, except at the tips, where they were bright red, almost the colour of blood. They were quite translucent, and presented an œdematous appearance. The symptoms were practically those of vesical calculus, and removal of the growth gave complete relief.

In the second case the lesion was very distinctly

FIG. 8.—View of the upper portion of urethra, showing a small mucous polypus in the lumen of the canal.

FIG. 9.—View of neck, which is deeply congested, and the lower surface is covered by a fringe of fimbriated processes with bulbous ends, pale and œdematous, except at the ends, which were bright-red and bled freely.

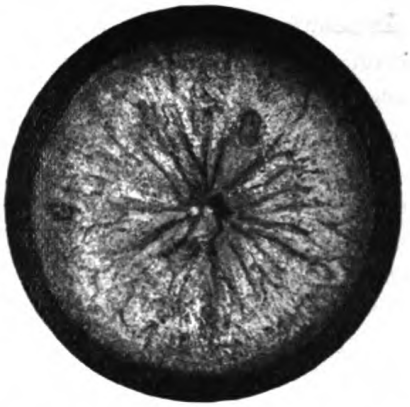

FIG. 10.—Chronic urethritis, with swelling of mucous membrane and numerous little nodular growths, oval in shape, and of a bright red colour. Form a mixed infection.

localised hyperæmia of the anterior wall of the bladder, immediately in front of the neck. The arteries were very numerous, and fully distended with bright blood, but there was no evidence of erosion or ulceration. This congested zone extended forwards about a quarter of an inch in some places, in others as far as half an inch from the neck of the bladder. It was associated with great pain, frequent micturition, and occasional hæmorrhage.

In the third case the lesions were little nodular growths, not larger than barleycorns, oval in shape, of a bright red colour, and protruding from the mucous membrane very much like the granulations in chronic pharyngitis (Fig. 10).

(F) Movable kidney may not only cause changes in the composition of the urine, such as the presence of albumen, blood, and tube-casts, and the accumulation of fatigue-toxins in the body, with diminution or increase in the secretion from time to time : but it may also cause reflex irritation, as shown by frequent and painful micturition. In a few instances frequency and pain in passing urine may be the only symptom complained of, and this being so the cause is very liable to escape notice, there being no symptoms directly pointing to the kidney. One peculiarity is that rest in bed at once affords relief, while active exercise causes the symptoms to return. Hence often the presence of calculus in the bladder is suspected.

CASE 8.—*Movable right kidney causing severe pain at end of micturition, probably by dragging on the right ureter when bladder was emptying itself—Nephrorrhaphy—Cure.*

Mrs. R—, æt. 34 years, was admitted to the Central Home on December 24th, 1905, complaining of severe pain at end of micturition, but at no other time, except on three occasions after travelling a considerable distance.

The pain complained of came on gradually during two years prior to admission, and for the last two months has caused great inconvenience and discomfort. Complete relief was obtained by rest in bed ; no pyuria or hæmaturia ; urine always normal, but sometimes very abundant.

Cystoscopic examination showed the bladder to be strictly normal ; no organisms present in the urine.

Abdominal examination showed the presence of a freely movable right kidney, which had not been suspected as no symptoms referred to the kidney. The patient was kept under observation for three months, when it was clearly found that there was no other condition present except the movable kidney to account for the symptoms. Rest in bed only gave relief for the time she was in bed ; as soon as she moved about the pain returned.

April 28th, 1906.—Nephrorrhaphy was performed, and after the operation the pain permanently disappeared.

The pain in this case was probably due to some dragging upon the right ureter when the bladder was emptying itself, there being no pain at other times.

June 30th, 1913.

ALIMENTARY INTOXICATION.*

By EDRED M. CORNER, M.B., M.C., F.R.C.S.
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GENTLEMEN,—I chose the subject of alimentary toxæmia for to-day because there has been much about it in the journals lately, and at the present moment there is a big discussion concerning it going on at the Royal Society of Medicine. I thought, therefore, that it might be of some use if we talked over the subject. To get at the bearings of what is called Alimentary Toxæmia, I must first explain that the poison is not taken by the mouth and absorbed by the alimentary canal, but is elaborated in the alimentary canal and absorbed therefrom.

It is best to start this subject from the beginning of life. As you know, a fœtus *in utero* has a certain amount of meconium in the alimentary tract. But there is no question of intestinal toxæmia from that meconium. The reason is that in the great majority of cases this meconium is sterile. But as soon as the baby is given any nourishment organisms are introduced into the alimentary tract. From that date what may be called its intestinal education begins. As life goes on the intestinal education continues, until we reach the final stage when all kinds of food which would be utter poison to the young, such as game, Stilton cheese, and other more or less decomposing foods, can be taken with equanimity.

One of the results of the introduction of micro-organisms into the alimentary canal of babes is this. If the child, as is so frequently the case, has a communication between the peritoneal cavity and the tunica vaginalis, it is very common for what is called a hydrocele to develop. That hydrocele, in the great majority of cases, is due to the fact that the organisms introduced into the intestine irritate the peritoneal covering of the bowel, and a certain amount of fluid is poured out into the peritoneal cavity. If that peritoneal cavity communicate with the tunica vaginalis a certain amount of fluid will percolate through into the tunica vaginalis, and so develop a hydrocele. That is not the invariable, but the usual cause of hydrocele in children.

* Delivered at the Polyclinic.

Later, if the child is not fed very carefully, and given unsuitable food, or improperly prepared food, or too much food, it develops rickets.

Another effect of this intestinal toxæmia, as seen in the very young, is the loss of fat and the wasting which these children frequently show. If the toxæmia is allowed to go on still longer, the child develops other illnesses which probably result from the toxæmia depreciating its powers of resistance.

With those sketchy remarks we can pass over the early years of childhood, and we come to the young adult. Then we find that this intestinal toxæmia, or intestinal sepsis, has had further effects. All these effects can be compared to the various chapters in a book; some are long and others are short. It is the same with the effects of this intestinal sepsis. One of the largest of these chapters of the results of intestinal sepsis in young adults is appendicitis. It is simply the result of organisms introduced into the alimentary canal. The great prevalence of appendicitis is really due to intestinal sepsis, which everybody born into the world must suffer from. In some patients the chapter, as we will call it, of appendicitis is a large one; in the majority of people it is a small one and not noticed.

But the appendix is not the only part of the bowel that gets irritated by this sepsis in children. They may have colitis; that is merely another result of sepsis.

Then, again, a very common thing found in children—in fact, one finds it in the abdomen of every child operated upon—is multiple glands in the mesentery. These glands are frequently tuberculous. That illustrates to you the fact that it is possible for the intestinal sepsis, by lowering the resistance, to allow of the admission of many organisms to the body. Of recent years we have learnt to recognise pneumococcal infections, and the statistics of the Registrar-General show that these infections, whether of the lungs, or of the joints, or elsewhere, are on the increase. That is another example of our greater powers of recognition, and it is highly probable that in a large number of these cases the organism enters the body from the alimentary tract.

One should mention yet another example of this intestinal sepsis seen in the young, and that is joint disease. We get a number of joint diseases which are commonly called osteo-arthritis. That osteo-

arthritis is probably the result of organisms which have gained admission to the body, through lowering the patient's power of resistance, by the alimentary canal.

Just recently, with the last month or so, I have come across another example of the migration of organisms from the alimentary canal into the joints. That was in a child who was admitted to Great Ormond Street with a suppurating shoulder. The shoulder-joint was opened, cultures were made from the pus, and to our great surprise we grew an organism which was very similar to the typhoid bacillus. This child had never had typhoid fever, and one made inquires about it, and found that albeit the majority of people might be hazy on the subject, there was a concensus of opinion that a number of the intestinal or alimentary ailments of children, although not genuine typhoid, were associated with the presence of the typhoid bacillus, or some organism very like it. That is another example of the infection of joints from the alimentary canal.

Passing from childhood, in after years one finds that the intestinal education has progressed considerably, and that what we may call the chapters in the book of intestinal education are much more numerous. We find that there are a number of infective conditions of the alimentary canal—and again I would remind you that these infective conditions are simply other chapters in the book of intestinal sepsis, and are comparable to appendicitis. Such an infective condition, for instance, is gastric ulcer. Quite recently a movement was set on foot, and the name coined, "appendix dyspepsia," that is, dyspepsia dependent on appendicitis. It was found that a number of patients who had had gastric ulcer and gastric dyspepsia for years could be completely cured by the simple operation of removing the appendix.

There is another class, namely, duodenal ulcer, which has fallen into the same category; and now a number of surgeons, when they do gastro-enterostomy for duodenal ulcer, always remove the appendix at the same time. The intimate association between affections of portions of the alimentary canal so far distant as the stomach and the duodenal and appendix region, shows that there must be some chain connecting these two situations. Our present knowledge suggests to us that this chain of infection is intestinal sepsis.

A further degree of this intestinal ulceration which may result from intestinal sepsis is seen in cases of ulcerative colitis. We know how difficult they are to treat, and how unsatisfactory they are to deal with in all ways. The reason is that when once the alimentary canal has become septic, it is very difficult indeed to render it sterile. Further, if we should render it sterile, or approximately sterile, then the patient who has been educated from birth upwards to a certain degree of intestinal toxæmia feels that if he does not have that degree of intestinal toxæmia, something is wanting and he is not well. For instance, X-rays inhibit, and even cause to die out, certain colonies of micro-organisms. When X-rays first came in, there was one man who had skiagraphs taken in all positions and conditions and, as the result of his body being penetrated by the X-rays for a long time, his alimentary canal became deficient in micro-organisms and his general health suffered. That was probably the first insatnce brought to our notice in proof of the fact that a certain amount of septic absorption from the alimentary canal is needful to our feeling of well-being.

It is obvious that in those situations where the material in the alimentary tract remains longest stationary the organisms will have a better chance of multiplying, and they will have the greatest chance of elaborating their toxins, and their results will be most potent. We are slowly beginning to recognise these things. Organisms can never make great headway in the stomach; owing to the presence of the acid medium, organisms will not grow well there. But when you get to the pylorus, then organisms may start. We find, for instance, a number of changes as the result of that. We get ulceration of the pylorus as a consequence of irritation of the pylorus. We get adhesions from chronic peritonitis around the pylorus. In the duodenum the contents flow through fairly quickly, so that the organisms are washed out. But again we find them about the junction of the duodenum with the jejunum, where the bowel takes a sharp kink, and certain organisms are left behind in what may be called backwater.

If we follow down the alimentary tract we find that through the jejunum and the upper part of the ileum the small intestine contents move quickly. As a result there is only a small growth of

organisms in those parts. But when you come to the lower part of the ileum the contents of the intestine move more slowly still, with the result that we again begin to get ulcerations and adhesions. When the contents of the alimentary canal reach the cæcum, the organisms spend a good long time there in an alkaline medium and in a warm place where there is plenty of food. As a result of all this growth of organisms in the cæcum we have appendicitis, ulcerations in the cæcum, and many adhesions, and the formation of what may be called new mesenteries. We find, therefore, that alimentary sepsis reaches its greatest degree in the cæcum, and then we see at once how it is that appendicitis is so common. From the cæcum onwards the intestinal contents begin to get drier from the absorption of water, and with the absorption of water the organisms do not grow so well. Hence the degree of alimentary sepsis gets less and less in the intestinal canal, from the cæcum downwards, and the effects of this intestinal sepsis are only present where the progress of the intestinal contents is impeded, such as in the flexures—the hepatic flexure, the splenic flexure, and the sigmoid flexure. In these situations you may have ulceration, and adhesions, and the formation of new mesenteries.

The slow passage and the drying of these intestinal contents introduces to you a subject which has come under notice of recent years, namely intestinal stasis. But there is at least one thing to guard us in connection with that. The great majority of examinations which are held to demonstrate or prove the existence of intestinal stasis are conducted by giving the patient a bismuth meal. The progress of that bismuth down the alimentary canal can be watched by means of X-ray examinations. As a result of these examinations we find that the bismuth will stay in the stomach some time, then it will travel slowly through the duodenum, then quickly through the jejunum and the upper part of the ileum; its passage will again become slow in the lower part of the ileum, then it will remain in the cæcum for a few hours; its passage can then be slowly noted down the colon, and still more slowly at the different flexures.

In conjunction with intestinal stasis must be taken into consideration the dryness of the intestinal contents. It is well known and well authenticated that people with intestinal stasis and dry fæces, commonly called constipation, can be perfectly well. I would direct your attention rather to the cases in which there is not so much constipation as moist fæces full of micro-organisms. You can also see, from what I have said, that amount of intestinal sepsis will exert its greatest effect from the lower part of the ileum downwards in the alimentary canal. You will also see that as everyone has to introduce food and micro-organisms into the alimentary canal, everyone runs a certain risk of intestinal sepsis, stasis, and toxæmia. But you will see that a certain amount is compatible with, or even necessary to, well-being, and that though everyone runs the risk, it is but few who suffer any marked effects.

Skin, as you know, is a tough structure, and does not absorb much, and therefore we contract comparatively few ills through our skin. But the mucosa of our alimentary canal is quite different; it has a highly absorbent surface, and everybody absorbs a certain amount of toxic products from the intestinal tract. This absorption of toxins from the intestinal tract starts practically at the moment of birth. In consequence, it has been said that it is through our alimentary tract that death begins to stalk us from the moment of birth; and that by lowering our resistance to organisms, or perhaps by the more direct access of toxic organisms from the alimentary canal, death may be hurried. In this connection Metchnikoff and others have shown that if only the alimentary canal can be rendered and maintained moderately aseptic, life may be prolonged. So that I would direct your attention to the fact that alimentary sepsis is a natural condition of all of us, and probably has a great deal to do with the length of life of the individual. We also see that such things as gastric ulcers, duodenal ulcers, colitis and appendicitis are mere chapters in the book of intestinal sepsis. We have further seen that mere intestinal stasis, without toxæmia, is of no great account.

There is just one other thing that we are apt to lose sight of, especially at the present day, namely, that our intestinal tract is not only a tract for absorption, but it is also a tract for excretion. Therefore, it might be argued that if only that tract be made perfect, our excretion of products will be good, and with such an excretion of products life might be rendered reasonably everlasting. But we

lose sight of the fact that our alimentary canal is not our only excretory organ. There are, in addition, our skin, and kidneys, and lungs. Therefore, no matter how good an intestinal tract we may have, its rectification need not necessarily improve our well-being. That we have had proved to us by such operations as removing the colon in short-circuit, which, although they do some people an immense amount of good, yet in other cases do no good, while in still others the patients seem to be worse off. The explanation is probably that we have not sufficient knowledge to enable us to differentiate which excretory system is deficient; and no matter how we may improve the excretory system that is not deficient, providing we leave the failing system alone, the patient is not improved.

There are two other subjects to which I would direct your attention. One is the use of purgatives. It is known with reasonable certainty that with the use of purgatives you have an increase in the number of flora—the organisms in the intestinal tract. The action of purgatives, as regards intestinal toxæmia, is purely temporary. When the purgative acts, the organisms are swept out of the alimentary canal; but if no more is done the organisms in the alimentary canal will probably be greater in number after the purgative than perhaps they were before, owing largely to the increased moisture present.

Then I would remind you that we have briefly gone over the onset of the various results of intestinal sepsis as we now know them. One started with the child and found comparatively few examples; then one found a greater number as the subjects grew older and older, until finally, perhaps, the effects of intestinal toxæmia increased so much in number that life ceased. Perchance in lowering the resistance of the body we have the genesis of such diseases as malignant disease. At present we do not know. But I would point out to you that if there is a question of doing anything in the surgical or medical way for intestinal sepsis or stasis, the day of doing something should not be postponed or delayed, because we know that the longer this treatment is delayed the more numerous and the greater will be the effects of the intestinal stasis and toxæmia. When once a patient suffering from intestinal toxæmia has to take the purgative treatment, that treatment ought to be prolonged, and not limited to a few occasional doses of the purgative.

In the brief time that we have had here to-day I have tried to give you a simple statement of the position of our knowledge of intestinal toxæmia at the present time. I hope you have seen from that statement that intestinal toxæmia is a condition which is as widespread as human life is, and that therefore everyone, in a more or less degree, must at times suffer from the result of it. Hence, you will see the importance of the subject, and in view of the prominence of the question to-day you will see the importance of appreciating the fact that a certain amount of intestinal toxæmia is natural, not only to the human race, but to all living animals.

June 30th, 1913.

Umbilical Concretions.—Herzenberg gives a short but monographic account of this very rare condition. He excludes from consideration all foreign bodies which have merely lodged temporarily in the fossa, without coincident change in the soft parts. The true concretions have the following genesis: The navel has a peculiar configuration, which favours the arrest of shreds of clothing, hairs, dirt, etc. A second factor is the presence of some tenacious secretion from the sebaceous glands, which acts as a cement. Upon this for a nucleus more sebum and epithelial *débris* are deposited until a conglomeration has formed. The presence of lime-salts makes the latter a sort of tophus which has received the name of umbilical calculus. Having attained a sort of natural limit of growth the concretion now begins to act as an irritant foreign body, setting up an inflammatory reaction which may involve a large area and simulate a tumour. Infection may readily occur with production of a phlegmon, the discharge from which contains in addition to pus crystals of cholesterin, fat drops and phosphates. As a rule from one to two months are required for the formation of one of these inflammatory tumours, this interval ending with suppuration. Given a phlegmon of the umbilical region, the presence in the discharge of phosphates points at once to the presence of a concretion. In the absence of phosphates in the discharge we have to think of the possibility of dermoid cysts, umbilical adenoma, and even of actinomycosis. In cases of alleged umbilical dermoid we should search for the presence of a concretion.—*Med. Record*, vol. lxxxiii, p. 1001.

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SOME CONDITIONS SIMULATING PROSTATIC HYPERTROPHY.*

By CUTHBERT WALLACE, F.R.C.S.,

Surgeon to St. Thomas's Hospital in charge of Out-patients.

THE treatment of the ordinary prostatic enlargement is now fairly well settled for the time being, and there is no difficulty in recommending a definite line. If the symptoms are well marked, if they interfere with the daily routine of the patient or limit his movement there is no hesitation in advising the removal of the obstructing growth, provided that the rectal examination shows distinct enlargement, which is definite in outline, bilobed and resilient to the touch. On the other hand, if the symptoms are not so prominent, and the organ not markedly enlarged, there is nothing to be lost by the exercise of a little patience. There can be no doubt that the adenomata do become more easily removable as they increase in size and age. Then there are cases which show a firm but moderate enlargement with but little rectal projection. Here the removal of the obstruction is likely to be difficult on account of the want of encapsulation, and should not be lightly undertaken. Then, again, patients of appropriate age present themselves for treatment with the usual symptoms of prostatic obstruction, in whom a rectal examination shows no enlargement or abnormality of the organ. In certain of these the cystoscope will show a gross intra-vesical growth, usually post-urethral. The disparity between the increase as shown by rectal examination and that found within the bladder is very remarkable. The following is a case in point:

CASE 1.—J. T. H—, a male, æt. 63 years, had for ten years suffered from frequency of micturition. For some weeks he had passed a catheter night and morning, there being residual urine to

* Lecture delivered at the Polyclinic.

the amount of ten ounces. The urine was acid. On rectal examination there was perhaps some slight enlargement of the prostate. On account of the early age at which the symptoms had commenced and of the negative examination of the rectum I was uncertain whether the symptoms were due to enlargement of the prostate or to some other cause. I suggested a cystoscopic examination with a view of clearing up the matter as far as possible, but I was not very much disappointed when the patient elected to go on as he was for a time. Three months later he came back and said that he must have relief. It was decided, therefore, to open the bladder and see what could be effected. Before this was done the cystoscope was passed, and it was noted that to get the beak to enter the bladder the eye-piece had to be very much depressed. On looking through the instrument it was evident that one had to do with a very big intra-vesical projection of the pre-spermatic portion of the prostate. Looking immediately over the top of the tumour the only thing visible was the fasciculated posterior wall of the bladder; the trigone was not visible. It was judged that the projection was one and a quarter inches high, and this was confirmed by comparing the different depth at which a light reflex was obtained according to whether the window of the cystoscope was looking forwards or backwards on its introduction. There was also an elevation on either side of the internal meatus. I show you the masses removed, of whose size the rectal examination gave no idea.

At another time the cystoscope shows only small bossy outgrowths about the internal meatus, suggesting submucous adenomata. These, again, are usually situated behind the urethra. Their removal is easy and is followed by a complete restoration of the bladder function. The insignificance of the tumours removed is remarkable, and points to the fact that a very small obstruction will cause very marked symptoms, as the following case will show.

Case 2.—J. W—, a male, æt. 69 years, had suffered from frequency of micturition for seven years. For the last five years he had passed the catheter three times a day to avoid the frequent calls to micturition. The urine was acid. The catheter used was a *coudé*. With a sound in the urethra only a veryslight enlargement of the middle lobe was thought to be present. The cystoscope showed a small post-urethral projection, but it was evidently insignificant from the fact that all the trigone was visible. The bladder was opened, and an inspection of the internal meatus showed that there was a very slight median projection behind the internal meatus. It was not more than a quarter of an inch high. It was quite sessile and could not have acted as a ball valve. A bimanual examination of the organ failed to demonstrate any other abnormality of the prostate. With the finger the small projection was removed, and with it came a strand of mucous membrane and fibrous tissue from the posterior urethral wall, which strand contained several minute adenomata. I could hardly have believed that so small a mass could have caused the symptoms, and was glad to find that when the bladder closed normal micturition returned and that the catheter was unnecessary.

In other cases the cystoscope reveals no abnormality of the prostate, but a bimanual examination through the open bladder shows small adenomata imbedded in the lateral lobes, the removal of which will render micturition normal.

There are many cases, however, in which all examination is negative with the exception of fasciculation of the bladder-wall. Under these conditions can the surgeon do anything to relieve the patient beyond seeing what a period of bladder drainage will effect? In this connection the following case is of interest.

Case 3.—J. W—, a male, æt. 80 years, was admitted with an over-flow retention. At first the case was considered an ordinary one of chronic retention due to prostatic enlargement. *Per rectum* there was no enlargement to be detected, and the cystoscope showed that there was no intra-vesical projection, the whole of the trigone being visible. There was well-marked coarse fasciculation of the muscle-fibre. When the bladder was opened a bimanual examination conducted with a sound in the urethra showed plainly that there was no adenomatous formation nor any substantial enlargement of the organ. The internal meatus was normal and, as is usual, admitted the tip of the index finger. There was no contraction or hardness of the tissues surrounding the entrance of the urethra. It was decided to see what a period of bladder drainage would do to restore the normal function, and with this intent a supra-pubic drain was established for some weeks. The tube was

then taken out and the bladder allowed to close, which it did after a considerable interval. The retention, however, continued, and the patient again became entirely dependent on the catheter. It did not seem that much could be done for the old man, but he was very anxious to be relieved of the catheter, and asked if nothing further could be effected.

The bladder was again opened and a wedge removed from the prostate immediately behind the urethra. The apex of the wedge lay about half an inch behind the urethra, and the base corresponded to the posterior wall of the urethra above the ejaculatory ducts. The tissue removed proved to be normal prostatic tissue.

The result of this procedure was good, and the patient left the hospital with only four ounces of residual urine, which quickly fell to one.

At first this case was thought to be one of mechanical obstruction due to enlarged prostate. When, however, the bimanual examination through the open bladder showed no abnormality of the prostate and a normal internal meatus, it was thought that the causation of the trouble must be due to a primary muscular atony or to some abnormal condition of the nervous mechanism with a consequent stretching and giving out of the muscle coats—a secondary atony. It was thought that rest would put the bladder right if the muscular failure was due to anything in the nature of "fatigue."

No improvement followed, so that it seemed that the muscle power was irretrievably lost or that the nervous arc was still at fault. It did not seem possible that the prostate was to be blamed, there was no enlargement or sign of chronic inflammation, and the internal meatus was normal.

Was the case to be labelled as one of "prostatism," that is, one in which the symptoms of prostatic enlargement are present without any anatomical alteration of the parts?

The success of the removal of a wedge of prostatic tissue in restoring normal micturition showed that the muscle and nervous elements were not to blame, and indicated that the symptoms were due to an anatomical defect. What was this defect?

In the normal bladder the prostatic urethra opens out of the middle of the lowest part of that organ. The entrance to the urethra is surrounded by the internal sphincter supported by the resilient tissue of the prostate. Under normal conditions this sphincter remains closed until the act of urination commences.

When hypertrophy of the prostate either of the adenomatous or fibrous type takes place, the disturbance of micturition is caused in two ways.

In the first place the action of the sphincter is disturbed by the growing gland, which first thins and then stretches it. In the second place—and it is the most important one for our subject—the urethra is distorted.

Owing to the fact that the increase in the organ takes place in the upper and pre-spermatic portion of the gland the urethra becomes bent in an antero-posterior direction. The apex of the angle is at the level of the ejaculatory ducts, and in some cases the upper and lower portions of the urethra make an angle of ninety degrees with one another. The internal meatus is at the same time carried forward so that it comes to lie at the bottom of the anterior bladder-wall. As is well known, it was to overcome this distortion that the *coudé* catheter was introduced.

It will be readily understood that this bending of the urethra will cause it to enter the bladder at an angle instead of in the usual vertical direction, so that the pressure of fluid on the trigone when the bladder begins to contract will force the upper wall of the urethra against the lower, thus forming a valve.

The opening of the urethra into the bladder will thus come to resemble the normal method by which the ureter enters the same viscus.

The greater the distortion of the urethra the thinner will be the layer of prostate between the trigone and the posterior urethral wall, and the more perfect the valve and the greater the effect of the bladder contractions in closing the canal.

The Mercier-Civiale-Guthrie-Bottini type of operation, which was very successful in many cases before the rise of the modern operation, produces destruction of that portion of the prostate which lies between the urethra and the trigone. The effect is to straighthen out the urethra, and it is to this the operation owes its success. The old procedure of tunnelling the prostrate had the same effect.

In like manner the enucleatio nof adenomatous masses in the modern operation of prostatectomy, by the removal of the pre-spermatic portion,

restores the normal axis of the bladder and urethra.

The operation in the case of the old man was the destruction of the pre-spermatic portion of the prostate, and its success suggests that the cause of the obstruction was of a similar nature to that just described—that is, to distortion of the uretha. Are there any facts that support this suggestion?

It has been pointed out—I believe by Thompson Walker in the first instance—that an antero-posterior bending of the urethra takes place in the prostates of men getting on in life without any increase in bulk. I have been able to confirm this observation in many instances, and have even marked the distortion in prostates below the normal size, and which might be described as in the state of atrophy. Unfortunately antero-posterior distortions of the urethra cannot be demonstrated by means of the X rays and a bismuth-filled catheter, as can lateral distortions, owing to the fact that, with the patient lying in the lateral position, the catheter is too far from the plate to show up.

If this is not the explanation it is difficult to account for the complete recovery that followed the destruction or division of the pre-spermatic portion of the organ. At all events it seems more in accordance with observed facts than any other explanation.

The lesson to be learned is that the retention in such cases can be relieved even though there is a failure to demonstrate any enlargement of the organ. The surgeon is thus relieved of the disappointment of having to close the bladder without making an attempt to cure the patient.

Case 4.—R. G—, a male, æt. 55 years, was admitted with a history of some years' frequency and difficulty of micturition. *Per rectum* the prostate was thought to be a little enlarged. There was residual urine to the amount of 10 oz. The cystoscope showed some slight fasciculation as the only abnormality. The urine was acid and clear. The bladder was opened above the pubes, and a bimanual examination showed that the prostate was not increased in size and that there was no intravesical projection. A wedge of prostatic tissue was removed from behind the urethra. The patient left the hospital with no residual urine and normal micturition.

This case is of the same nature as the last, but the symptoms less marked. This patient had frequency and only a partial retention, but there was no obvious cause for the retention, and the operative procedure was similar, both in execution and in the result. It is to be noted that the age is considerably less.

The next case would perhaps be placed in a different category to the two preceding ones on account of the comparative early age at which the symptoms began, and the possibility of prostatic enlargement might hardly arise in the attendant's mind.

Case 5.—J. L—, a male, æt. 49 years, had suffered from difficulty and frequency for four years. Nine months before he came under treatment it was found that there were 9 oz. of residual urine. When examined the residual urine had increased to 14 oz. A rectal examination with a sound in the urethra failed to show any alteration in the size or consistence of the prostate. There was no sign of prostatitis. The urine was clear and acid. The cystoscope showed only a moderate fasciculation of the bladder-muscle. There was no alteration in the trigonal area of the bladder. A large steel sound passed easily. The catheter was resorted to night and morning to avoid the frequent calls to micturition. He had seen many surgeons. One had advised immediate operation for carcinoma but the others advised him to go on as he was. There was no sign of nervous disease.

Here was a man, æt. 49 years, who had for four years suffered from difficulty and frequency of micturition, and who had had, for at least nine months, 9 oz. of residual urine.

This case is by no means singular, and the symptoms may begin even earlier. These cases have always been a puzzle, and many explanations have been offered and much written about them.

It is to this class of case especially that the term "vesical prostatism" has been applied. The group is very ill defined; it might almost be described as a rubbish-heap on to which is thrown all those cases presenting difficulty and frequency of micturition in which the medical attendant can find no adequate explanation.

The following defects have from time to time been advanced to explain the symptoms: (1) Induration of the internal meatus. (2) A primary atony of the bladder muscle. (3) A secondary failure of the muscle due to inflammation. (4) A fibrosis of

the muscle due to old age. (5) A want of correlation between the sphincter and the detrusor. (6) A loss of sensibility of the mucous membrane. (7) Chronic prostatitis and post-urethral bar formation. (8) An atrophy of the prostate. (9) Perverted prostatic secretion acting on the bladder. (10) Failure of the extra-spinal bladder centre.

In the present instance there were no signs of prostatitis or of a present or previous cystitis or of the post-urethral bar. Atrophy of the prostate and senile fibrosis of the muscle (if such a condition does really exist) can be excluded on account of the age of the patient.

The ease with which a large sound passed negatived any induration of the internal meatus.

If the other suggested causes are examined they, too, do not seem adequate to explain all the facts.

A want of correlation between the sphincter and the detrusor muscle will explain stammering or intermittent micturition, but it does not seem capable of accounting for a complete retention or for the bladder expelling a certain amount of urine and then refusing to complete its task. It is remarkable how constant is the amount of residual urine—it seldom varies more than an ounce or two. The detrusor always contracts to a certain point and then ceases. If a want of agreement between the sphincter and detrusor were the cause one would expect the amount of residual urine to vary considerably from time to time. A nervous paralysis of the detrusor such as occurs in the reflex post-operative retention is, for the same reason, unable to offer a solution.

It has been suggested that some impairment of sensation may be at the bottom of these cases. Mr. Shattock has shown that when the mucous membrane is rendered insensitive by cocaine that the bladder of cats dilates without any attempt at expulsion. Here again we have the difficulty that the bladder "in prostatism," after some hesitation it may be, acts for a time and then fails before its work is done. A complete retention is easy of explanation, an incomplete one is not.

Again, intact sensibility is only required to start the act. If this were not the case a normal individual would be constantly wanting to micturate before the bladder had become at all full. This of course does happen when the mucous membrane is inflamed and frequent micturition is the result. Once started the act should be completed without the need of further impulses from the mucous membrane.

In this particular class of case the act is started but not finished. It is the failure to complete, not to start the act, that requires explanation.

The condition is best described by saying that the detrusor is in such a state that it will contract up to a certain point and then ceases to act. It would almost appear as if it soon tired and had not time to recover before it was again called upon to act, or that its fibres for some reason could only shorten to a certain degree. Even if there is no satisfactory explanation, mechanical or otherwise, of the behaviour of the detrusor in cases of residual urine, the important fact remains that retention or the residual urine found in enlarged prostate which is admittedly due to a mechanical obstruction disappears when the obstruction is removed. The case of the old man, æt. 80 years (Case 3), and of R. G—. æt. 55 years (Case 4), shows that such an obstruction can exist without its presence being easy of detection. It was pointed out that a distortion of the urethra was a probable cause. Can such a distortion be present in some cases of "vesical prostatism"? If so, we have a method of relieving such patients of the need of catheterisation and its attendant evils.

It is interesting that in chronic prostatitis, which was advanced as a cause of prostatism, the residual urine has been shown to be due to a mechanical cause This disease has attracted a good deal more attention abroad and in America than in this country. It is not necessary to discuss here whether all the symptoms and physical signs attributed to the disease have their origin in the inflammation of the prostate. It is sufficient for my purpose that the disease may have to be distinguished from prostatic hypertrophy, and that one of its symptoms is relieved by a special procedure.

The patients with this disease suffer from various sexual disturbances and from referred pains about the genitals, buttocks and thighs. To a rectal examination the prostate may be normal or a little firmer and larger than normal. The prostatic secretion obtained by massage contains pus-cells. And it is on the presence of these pus-cells that the diagnosis mostly rests. The urinary symptoms are difficult, and frequent micturition accompanied by (what is of interest here) residual urine or a complete retention. Young, of Baltimore, who has

written greatly on this subject, says that many of the cases had symptoms typical of prostatic hypertrophy, and that he was surprised to find no enlargement of the organ. The age of the patients was commonly between fifty and sixty years, but many are younger. The cystoscope shows signs of obstruction—trabeculation of the bladder-wall accompanied with hypertrophy. The obstruction to micturition is attributed to a post-urethral bar which shows up as a slight elevation of the trigone, and is itself said to be due to some thickening of the pre-spermatic portion of the prostate, as evidenced by a bimanual examination with a sound or a cystoscope in the urethra.

The cases were cured by punching away the pre-spermatic portion of the prostate by a special instrument introduced into the urethra.

It is interesting to note that this bar at the neck of the bladder, to which Young attributes the residual urine in chronic prostatitis, was first described by Guthrie in 1836. Much has also been written on the same subject by Civiale, Mercier and other French surgeons, who devised instruments for its division. In some cases the treatment was successful, but in many it failed. This is not to be wondered at, as many cases of prostatic hypertrophy were included in those so treated. Sir Henry Thompson, who reviewed the whole subject in his book, came to the following conclusions about its causation :

(1) That in the majority of cases the bar was an outgrowth from the pre-spermatic portion of the prostate.

(2) That an organic obstruction may exist at the neck of the bladder where there is no enlargement of the pre-spermatic portion of the gland.

(3) That in some cases it is due to an hypertrophy of the muscle-fibres of the neck brought about by stricture, calculus, and occasionally without any of these conditions.

(4) That sometimes the obstruction is due to a fold of mucous membrane raised by the projection into the bladder of the lateral lobes of the prostate.

I have never seen a proper explanation of how the bar can cause obstruction to micturition. As described by different observers from the time of Guthrie to the present date, it seems totally inadequate to produce the symptoms attributed to it. I am tempted to suggest that the true cause of the obstruction is the bending of the urethra within the prostate.

Marion, the French surgeon, has recently published a paper, in which he quoted cases in which very insignificant submucous adenomata which had escaped detection until the bladder had been opened had caused retention of urine. In one case the cause of the retention was a small papilloma of the prostatic urethra. In another a thickening of the neck of the bladder was the apparent cause, for the retention was relieved when this cause was removed. He finished the paper with the opinion that the diagnosis " vesical prostatism," that is, of the syndrome of prostatic hypertrophy without enlargement of that organ, should be taken with great reserve. This confirms me in my opinion that one should not hesitate to open the bladder of a patient with symptoms of prostatic hypertrophy, although clinical examination has failed to show any cause for the obstruction.

It is to be noted that the phenomenon of residual urine is very seldom found in women. If the bladder was the organ at fault, the two sexes should be affected in like proportion. The disparity of the frequency of the affection in the sexes suggests that the cause is to be found in some difference in the urethra. Suspicion thus at once falls on the prostate, which is alone found in the male.

Even if many cases that would appear to be due to a fault of the muscle or nerve supply of the bladder turn out to be, after all, due to a mechanical defect of the urethra, there are still cases which it is not possible at the present time to account for in this way. There are many well-observed cases in which the phenomenon of residual urine combined with frequency and difficulty of micturition commences at a comparatively early age, and it is impossible to deny that some may persist until the age at which they come under observation may raise the suspicion of prostatic enlargement. The causation of such cases is unknown. A good deal will depend on the view that is taken of the causation of fasciculation of the bladder. Is it to be considered as the evidence of an obstruction, or can it be found without a mechanical cause? Opinions differ on this subject, and I may refer you to a paper read in this place by Mr. Thompson Walker, who has given reasons for believing that one type of fasciculation is not caused by obstruction.

Idiopathic hypertrophy and dilatation of other viscera are well known, and these conditions have been regarded as analogous to the bladder in the state under discussion. Those affected are the œsophagus, the stomach, and the colon. I would refer you to the paper on this subject by Mr. Shattock in the 'Proceedings of the Royal Society of Medicine,' 1909. Dilatation of the colon and of the œsophagus seem to resemble idiopathic dilatation of the bladder more closely, as there is no obvious cause, while in that of the stomach there is the well-marked thickening of the pyloric sphincter; no one has described such a condition of the vesical sphincter.

If it be granted that residual urine can exist without mechanical obstruction, I would suggest that the state of the bladder is nearer that of the large intestine in chronic constipation than to that of organs suffering from idiopathic dilatations.

In chronic constipation the bowel gets rid—it may be with great regularity every morning—of fæces that it should have passed twenty-four or forty-eight hours earlier. In the same way the bladder acts regularly, if a little more frequently than normal, but never rids itself of its contents. In both cases very likely the patient is quite unaware that there is anything amiss with the particular organ. Of course the differences in the consistency of the contents and their modes of delivery to the anus and urethra respectively makes a complete comparison impossible. If the urine of man was solid, as it is in birds, and if the ureters opened at the top of the bladder, the condition of the urinary bladder with an incomplete retention and of the rectum in chronic constipation would be almost exactly similar.

Vesical calculus is credited with producing difficult and intermittent micturition together with residual urine. A hypertrophy of the trigone muscle is said to be the cause of the errors of micturition. If this is the case a condition of things will arise which has to be distinguished from enlarged prostate and from "vesical prostatism."

In the young, vesical calculus gives rise to very definite symptoms, mostly caused by irritation of the mucous membrane. In the aged the painful symptoms do not show themselves, so that without the cystoscope or sound vesical calculus may not be diagnosed. I will conclude this lecture with relating a case.

D. G—, a male, æt. 65 years. Had suffered from frequent, delayed and interrupted micturition for five weeks. During the night the bladder was emptied four times. There was no pain or discomfort. There were 10 oz. of residual urine. *Per rectum* the prostate was normal. The urine was acid.

The cystoscope showed that the bladder mucous membrane was inflamed, and that the muscle-fibre was hypertrophied. There was a post-trigonal pouch, which was full of small calculi. There was no intra-vesical projection of the prostate. The bladder was opened above the pubes, and about a score of soft cod's-roe stones were removed. They were composed of phosphate and carbonate of lime with some uric acid.

Bimanually the prostate was quite normal.

In due course the bladder closed, and normal micturition re-established. A month later there was still 7 oz. of residual urine.

In this case the symptoms, mainly on account of the absence of pain and the presence of residual urine, suggested enlargement of the prostate. When the rectal examination turned out negative there was still the possibility of an intra-vesical enlargement.

The cystoscope showed that there were many stones, but no intra-vesical enlargement. There might still have been some enlargement that only a bimanual examination could detect. On account of the nature of the stones there was no question of using a lithotrite, so the bladder was opened, and any enlargement of the prostate was negatived by the bimanual examination. The internal meatus was normal, nor was there any bar formation.

The question had to be decided as to whether it was best to perform a prostatotomy or simply to remove the calculi, and trust to their having been the cause of the trouble. I decided on the latter course. The residual urine has fallen to 7 oz. since the bladder closed six weeks ago. Time will show if the calculi were the cause of the trouble or whether it would have been better to have performed a prostatotomy. At present the man is unaware that there is anything wrong, as the amount of residual urine is not enough to cause frequency, and his difficulty has disappeared.

Conclusions.

(1) That many cases present symptoms which at first sight might reasonably be considered as due to prostatic hypertrophy, but which subsequent examination proves to be due to other causes.

(2) That prostatic enlargement can only be excluded by a bimanual examination through the opened bladder.

(3) That even when a bimanual examination shows no enlargement to exist the cause of the errors of micturition may still lie within the prostate.

(4) That no error of micturition should be assigned to a failure of nerve or muscle until all mechanical defects have been excluded.

(5) That a bending of the prostatic urethra is the cause of the difficult micturition, in some cases at all events, and the patient can be cured by a simple operation.

July 7th, 1913.

Tuberculous Dactylitis.—R. M. Green concludes that tuberculous osteomyelitis of the digits is essentially a disease of infancy and childhood presenting a definite clinical picture and tending strongly to spontaneous recovery. Pathologically it is characterised by bone destruction and rarefaction and by chronic inflammatory reaction in the surrounding soft tissues. It may heal by resolution and absorption, or by sequestration, suppuration, and sinus formation. Its differential diagnosis from pyogenic osteomyelitis, sarcoma, and syphilitic dactylitis may be made by clinical signs and X-ray examination. Its prognosis for anatomical and functional recovery is good and is proportionate to the amount of bone destruction and scar-formation. Therapeutically primary emphasis is to be laid upon general hygiene and local fixation. Incision should rarely be resorted to, since it complicates the dressing, protracts convalescence, and increases subsequent deformity and disability. Amputation is almost never indicated. Probably treatment with the X rays, or other forms of irradiation, is a valuable adjunct to the conservative therapeutic measures above described, since it expedites recovery by stimulating natural reparative processes. —*Med. Record*, vol. lxxxiii, p. 1089.

A LECTURE
ON
COLOUR-VISION AND COLOUR-BLINDNESS, AND METHODS OF TESTING.

Delivered at the Royal London Ophthalmic Hospital, City Road, on Monday, May 13th, 1913.

By C. DEVEREUX MARSHALL, F.R.C.S.,
Surgeon to the Hospital.

LADIES AND GENTLEMEN,—I want to go over with you to-day the question of colour-vision, colour-blindness, and methods of testing for these conditions, with the idea of helping you to come to a conclusion, in your own minds, as to whether the person you are examining is or is not colour-blind? That is really the important thing at which one aims, and many of the tests which are in general use are singularly deficient in this respect.

First of all it is necessary that I enter a little into the theory of the subject. The two theories which are most talked about at the present day—though there are others—are the Young-Helmholtz on the one hand, and that propounded by Edridge-Green on the other.

The Young-Helmholtz theory, as you perhaps remember from your student days, is based upon the conception that in the retina are supposed to be certain elements, the stimulation of which produce certain colours, one set producing a red curve, another a green curve, and another a violet, and that by the admixture of these all visible colours result. That is to say, if you stimulate the red elements you see a red colour; if you stimulate the area common to red and green, the resulting colour seen will be yellow. It is said that according to the part stimulated so you will get any colour pure, or any shade of colour. Mixed with the green, for instance, there may be some violet, and so you get blue. These elements, then, are supposed to exist in the retina. I say "supposed to exist," because no one has ever seen them or been able to demonstrate them. If you take the eye of a colour-blind person, you find no variation in structure whatever from that seen in the eye of the person with the full range of colour-vision, whether the observation be made during life or after death. The Young-Helmholtz

theory is held by a certain number of people, but they are for the most part physicists and not physiologists. On this theory is based one of the usual tests for colour-blindness, namely, Holmgren's wools. I show them to you. They consist of three test-colours—one light green, one red, and the other purple—and the person being examined is told to pick out certain colours which match the test or as nearly so as possible. I shall not at the moment go into the question of how the testing is done, but the test is founded on the Helmholtz theory, but all the known facts of colour-vision go to prove its fallacy.

The next theory, and the only one which accounts for the facts found to be associated with colour-blindness, is that of Edridge-Green, which is a different conception altogether. What he says is this: That colour-vision is not a special function of the eye at all, but one which is performed by the brain; that there is a certain centre in the brain which is stimulated by colours, and that this centre may be developed either highly or only rudimentarily. If it scarcely exists, there is total colour-blindness, associated with a structurally complete eye. If the centre is in a rudimentary state, the person can see red at one end of the spectrum and violet at the other end; he has no perception of colours which are intermediate in the spectrum, except by some difference in illumination or in shade. In a person with the visual centre developed still more there is a perception of more colours. You will remember you can have no conception or perception of colour except in the presence of light; no body possesses such a thing as colour in itself. If a body which is red be taken into a dark room, its colour is gone. Why has that body got colour when seen by the aid of light? Because it has the property of absorbing all the rays of light which impinge on it except the red rays, and they are reflected or turned back to the eye; and the same in the case of a body, say, of blue colour; it absorbs all the rays of the spectrum except the blue, hence it is seen as blue. Of course white light is a compound of all the colours of the spectrum, and if you separate one or more, the residue is not white, but something else. If we take a person whose brain in this respect is very primitive, that is, who has no developed colour sense, then to him the spectrum appears merely as an uniform grey streak of light, having no colour, though he might recognise that one part of the spectrum was brighter than another. Such a person is spoken of as having monochromatic vision. Though, as you know, the wave lengths of light at the red end differ materially from those at the violet end, such a person is incapable of telling the difference. It is a comparable case to that of the person who is said to have "no ear for music," who could not remember or reproduce a tune he had heard, and who, though possibly able to tell the difference between a bass and a treble note, could not distinguish between two notes anywhere near each other on the scale. Our next stage of development after the monochromatic is the person who sees a patch of red at one end of the spectrum and a patch of violet at the other, and the intervening space appears as grey. This person is known as a dichromic. That is a form which is commonly met with at the present time. In the next stage of development the person sees red at one end and violet at the other, but with practically no neutral band between. So you may encounter a dichromic person either with or without a neutral band. As development goes on the person still sees red at one end and violet at the other, but he sees a colour between, which is green. Thus you have the trichromic case, in which red, green and violet are perceived. You see that as more colours are crowded into the same space the area occupied by each must be less; hence the red in the trichromic case is not so extensive as in the dichromic. A dichromic person often says he sees green in the centre when he does not; in reality he will name green anything which is neutral to him. As I try to indicate what takes place at each successive stage of development I want you to notice that the new colour which the further development enables the person to see always comes where the difference in wave-length is greatest. In the next stage the examinee sees red, violet and green and yellow and the latter colour is found where the greatest difference of wave-length exists; here is our four-unit person or tetrachromic. Notice how much shorter the red and violet are getting; they are being pushed further apart. So that what the dichromic would call red, the tetrachromic would call yellow. The next stage is the person who also sees the same colours but one more in addition. The next greatest distance will be between the green and the violet, and so the fifth colour per-

ceived by our more developed centre is blue; these are the pentachromics. The next colour to be developed is orange, so that this person has a six-colour spectrum, and that is the colour range of the vast majority of people, namely the capacity to see in the spectrum red, orange, yellow, green, blue and violet. Such persons are called hexachromics. If development goes a stage further, a seventh colour will slip in—indigo—between the blue and the violet. That is the highest development of colour-vision there is, and such a person can perceive extraordinarily fine differences of colour. But remember no one of the groups of persons I have described will see the spectrum exactly like any of the other groups. If one person has to get seven colours into the spectrum in the same space as another has to get four, what one calls violet another will call blue. There are very few people who distinguish indigo as a separate colour at all; I do not mean who can pick it out of a paint-box, but in whose spectrum indigo has a place. What you need to do to test this is to have a pointer and have the exact dividing line between the colours indicated; you will find there will be differences of opinion as to the point of junction. The majority of men who have six colours in their spectrum will not agree with the marking off of the spectrum by others who have more or less; but there is only one person in several thousands who can see the whole seven colours.

That is a very reasonable theory of colour vision, and explains what is actually observed. Why should there not be different degrees of development of the colour sense just as there are differences in musical perception and ability? For both, in order to have the necessary perception and conception, the centres concerned must be present. For instance you cannot possibly train musically a person who cannot recognise the National Anthem being played, except by seeing people rise and the men take off their hats. We know there are servants who have to answer half-a-dozen different bells, each with a slightly different tone from the others, yet who cannot tell one sound from the other, and have to look each time to see which bell is moving. So also it is of no use trying to make these colour-blind or colour-defective people tell colours like others in whom the centre is properly developed. However good otherwise, and however scientific a person may be, if he has grown up without colour vision developed, he is not going to acquire it. Dalton, the scientist, was colour-blind, but Sir Isaac Newton described indigo in the spectrum; he was one of the few who was able to see indigo.

Now the practical object in examining colour-vision is to separate the dangerously colour-blind from the others. What constitutes the dangerously colour-blind person? The dangerous colour-blinds are those belonging to the first, second and third grades of those I have been describing. The monochromics are, of course, obviously colour-blind. I have seen only one instance of that, and I do not expect to ever see another; they are very rare. The second class, or dichromics, are the ordinary colour-blinds, while the trichromics are frequently in difficulties with yellow. In this matter you have to take into consideration what it is the person will be required to do. The look-out at sea and the railway-train driver always has to know a red and green light when he sees it under any conceivable circumstances, and also to know the difference between either, and a yellow light; he must have no hesitation or doubt in his mind whenever he sees any colour which is used in signals. No one will concern himself whether a driver distinguishes violet from blue, because such distinctions are not needed for signals; there is no regulation which says a blue light is safe and a violet light is dangerous. The colours most used for signal lights are red, yellow, and green, the yellow, of course, being what is usually termed white. In these circumstances you must settle on some test by which you can distinguish one from the other. How shall you do that? It can be done either by means of lights, or with coloured fabrics.

The ordinary test which is in vogue—though I cannot tell you why, because it is the worst of all the tests—is what is known as Holmgren's test; it is a wool-matching test. It is based on the idea that if a person does not see his colours he cannot match them. But that is not actually so at all. Let me explain why. Take a person such as I have described, whose spectrum consists of three colours—red, green, violet. If he knows that red is red, that green is green, and that violet is violet, he will know that blue is not quite the same as violet, and so he will tell you there is a difference between them; yet he will never know a blue light as blue when he sees it. He does not recognise

yellow as such, but he knows that what you call yellow does not look quite like either red or green, and so he will perhaps call it yellow, but will usually describe it as a sort of reddish-green. Such a person will be too sharp to pick up all the confusion colours and put them together. He knows very well, from his own observations, that he is capable of seeing certain colours, and he also knows there are certain colours which he is in considerable doubt over. But all he has to do is to get a normal-sighted person to come along and tell him what are the colours he is doubtful about; and although he may not be able to tell the difference between certain colours, or to name them, he will not allow himself to be caught matching them. He will have sufficient memory for a short time after a lesson to tell him he must not match things which have to him certain differences of shade and saturation. And you must remember a very peculiar condition which is attached to the Holmgren test, that is, that under no conditions is the examinee to be asked to name a colour. The test is bad enough in all conscience, but with this added condition it is quite impracticable, and you will be passing half the colour-blind people as normal whom you test by it. If you take up this grey skein of wool the colour-blind person would probably say it is green, but he will not put it with the green wools when he has the green with which to compare it. I hold that a test for colour vision is not worth anything unless you use names. It is said by some that the person being examined should not be asked to name colours because he may not know the names of the colours. But I say that if a person sees red and has not learnt to call it red, and sees green as green and does not know it as green, the sooner he goes away and learns the names of the colours the better. Such colour-ignorance is non-existent. The only reason a person does not speak of red as red, or of green as green, is that he does not see them as red and as green respectively. If a person has sufficient intelligence and education to grow up as an ordinary member of society and present himself to the Board of Trade or any other body as a candidate for a post, his failure to name green as green must be because he does not see it as green, and he is colour-blind. So you see any test in which you are forbidden to use names must be of very small value. The only advantage which the test by wools has is that the wools are cheap. But they soon get dirty, and, moreover, anybody can buy wools, and there is nothing to prevent a normal-sighted person coaching a colour-blind individual, and if it is only a question of matching, most of them will pass. A large proportion of colour-matchers in the various establishments are colour-blind people; they have such a carefully educated sense of shade-perception that they are quite successful matchers; so that even though they may not recognise the colours, and could not distinguish a separate colour if seen alone, they match extremely well. Some of them come within the category of dangerously colour-blind.

How else, then, shall you test? As I pointed out before, you do not want to reject everybody who has any colour defect; you only need to reject persons who are dangerously colour-blind, and to distinguish carefully between those who would be a danger if employed on railways or at sea and the others. You do not need to pay any attention to whether the fine differences which the draper talks about, such as puce and magenta, can be perceived by the candidate, nor whether he knows "crushed strawberry" colour or "electric blue." A seven-, six- or five-unit person does not come into the danger category. You want to assure yourself that the person whom you pass shall never have the slightest doubt or hesitation what colour a signal light may be which is suddenly projected into his vision when there is nothing at hand with which to compare it. The intelligent dichromic person will probably tell you the coloured fabrics quite easily. He will tell you green every time, but not because he sees it as green, but because he assumes that his neutral patch between red and violet is green; but if you give him a yellow substance he may still call it green, and if it be nearer the red end he will call it red. But you have to consider the case of the sailor who suddenly sees a light loom up in front of him, and, without anything to compare it with, has to know the colour of it. With such a person as we are considering, the colour he will judge a light to be greatly depends on the brightness of it—that is to say, if a light is a good way off, he may adjudge it to be a different colour from what he would call the same light when seen near. You have only to remember that vessels are navigated by lights to appreciate the importance of this matter. A man may be on the look-out, when

it is his duty to report what he sees to the bridge, and the helm is shifted accordingly. If the man says what he sees is a red light when it is a yellow or green one, you will see how easy a collision may occur. Remember, then, that the three-unit people are very dangerous, because their defect is very difficult to spot; they will see red, violet and green, but will be in confusion as to yellow and blue, and if you show them a yellow light and they do not know what it is, they will probably say it is red at one moment and green the next. Suppose a person sees a "bright," *i. e.* white, light showing, and calls it red, or he calls it green. If it is "bright," it is probably carried by a vessel lying at anchor; if it is red or green, it is a vessel under way. If to such a person you throw up a light of unknown brilliancy, he will be in doubt as to whether it is red or green, or he may correctly call it yellow. If a person is quite sure when he sees red, yellow and green under any condition, you do not need to reject him, because those are the usual signal colours; it is not material to his work that he cannot distinguish orange or blue.

I have condemned the Holmgren test roundly, and it is really too bad to justify you in giving it house-room. But what about the others? The best test certainly is the lantern test. Here I show you the Edridge-Green colour lamp. There is an electric light in this cylinder, and the light is projected through glasses of different colours to the eye. The first glass gives you yellow, the second green, the third signal green, the fourth blue, the fifth purple, the sixth gives red No. 1, the seventh red No. 2. There are three discs which show exactly the same colours, and you can mix any three in the beam which reaches the eye of the examinee. The most important point is that there is a fourth disc, which modifies any light which you throw up. There is a ground glass, a ribbed glass, and a neutral No. 1, No. 2, No. 3, No. 4, No. 5, so that you can reduce any light to extinction. There is no person I have ever come across or heard about who could pass this test who had not normal acuity of colour vision. With three discs, each containing seven colours and seven modifying glasses, the number of combinations you can get is infinite, and the person being examined can never know what you are going to do. If you have before you a two- or three-unit person, and you show him a green light which he may name correctly, and if then you slip in a neutral glass modifying the intensity of light, he thinks you have altered the colour and probably names it differently in accordance with that belief, and so when its brilliancy is diminished he may call it red. One mistake like that ought to ensure rejection at once. There is no colour-blind person, no matter how well educated, who can pass this test. I know this instrument through and through, yet if my colour vision were defective I could easily be caught out, because I should never know what colour was coming up next. A dichromic, if shown a red light through a wide aperture, will probably say it is red, whereas if you show the same colour through a small aperture he may say it is green or white. You should reject such a person at once. Another thing about the instrument and the test is that it is eminently satisfactory to the patient, for he has only to bring a friend in, and that friend if of normal colour vision will at once see when mistakes are made. This, then, is one of the most satisfactory tests that have ever been introduced.

You may ask, "Why not use a spectroscope?" So you might if you were able to spend two or three hours over every case, and though the Edridge-Green spectrometer is a lovely instrument, it costs about fifty guineas.

Here, in this box, is another testing method, and it is useful in confirming impressions. It is the Edridge-Green wool test. Here there are many more wools and a greater range of colours than in the Holmgren. You pick out an orange skein and ask the patient to name it and to pick out other orange ones. You will probably find him picking up the yellow ones and perhaps other colours also as matches for the orange. In the same way you test him with other colours. Then here are some silks. There are people who have no trouble with the colours of wools, but are in difficulties with shiny things like silks. A skein of slate-coloured silk will perhaps be regarded as a definite colour. Salmon-pink is another colour with which candidates with defective colour-vision get into difficulties, for to them it appears like two or three colours and they do not know which. There are also pieces of coloured glass—which can be kept clean—and coloured cards. But none of these are as good as the lantern.

Here is another very nice test, which likewise is Edridge-Green's. It is a box in which are placed beads of various colours, and in the lid are compartments labelled with the primary colours with

small holes to admit the beads. The person is provided with a pair of forceps and asked to place the beads in the suitable compartments. Some of the beads have shiny surfaces, others have dull ones, and when once the beads have been allocated they cannot be seen again until the diaphragms are removed and the mistakes are standing evidence against him. So here there is no question of matching; the beads are placed where they are supposed to belong.

There is also another very good test which I must mention, though the disadvantage of it is that anyone can learn it. It is called Stilling's plates.

Do not be led into the belief that a lantern which shows two lights is of value, because then the person being examined will compare one light with the other and may not be caught at all. But if you can throw a light of unknown intensity, the colour-blind will always be in difficulties with it.

There is one dangerous class of colour-blind person who has a shortening of the red end of the spectrum; they are the people who see very little red; the best of us can only see red of a certain intensity; beyond that there are many rays which the eye cannot see. But we want some assurance that a person who is to be a signalman can see red so long as it is to be seen by normal-sighted persons. If they cannot do so, they are dangerous people to have in connection with signals. These dark red rays are the most penetrating rays in a fog and may be the only ones to get through, but if the man has a shortened red end he will not perceive them, and an engine-driver will drive his train past such a signal-light. Put a red light in your lantern, then place a neutral before it to darken it, and ask the candidate what it is; if he has a shortened red end he will not perceive it at all, though it is quite visible to a person of normal colour-vision. These latter people cannot be discovered with the Holmgren test. The so-called red-blind person is a dichromic who has a shortened red end of the spectrum. The person to whom the violet end is shortened is not so dangerous, because violet lights are not used for signals. A good deal of objection has been urged against the use of electric lights for lighthouses, because even a powerful light cannot be discerned through a fog. That is because in that light there is a deficiency of red rays which penetrate fogs.

I would therefore say you must be very careful in testing these people; they are often very difficult to discover, but if you will accept the proper theory, the one which I have told you permits of an explanation of the observed facts, you can classify your cases; and the use of the lantern of Edridge-Green, or failing that, one of his other tests, will enable you to separate dangerously colour-blind persons from those who, from this standpoint, may be considered safe.

July 7th, 1913.

TRAUMATIC PANCREATIC PSEUDO-CYST.*

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In 1892 Jordan Lloyd contributed an important paper on the subject of pancreatic cysts to the *British Medical Journal*. The main objects of his paper were (1) to establish the direct causal relationship between traumata of the upper abdomen and the development of cystic swellings in the neighbourhood of the pancreas, and (2) to prove that these traumatic cysts are in many cases accumulations of fluid in the lesser sac of the peritoneum rather than true retention cysts of the pancreas. Both of these contentions have received general acceptance, and it is now customary to speak of such cystic developments as pancreatic pseudo-cysts to distinguish them from the numerous varieties of true pancreatic cysts. The most generally received view of their formation assumes that, as the result of an injury to the pancreas, blood and pancreatic secretion are poured into the lesser sac, and that as a result of the low-grade inflammation thereby established the foramen of Winslow becomes occluded, so that increasing distension of the sac from accumulating pancreatic secretion becomes possible. The formation of cysts of this sort is usually brought about within a comparatively short period after the causal injury, and therein lies one of the more important means of diagnosticating pseudo-cysts. Occasionally, years after an injury a cyst may develop, but, according to Körte, in such a case the development is of a true retention cyst nature secondary to a localised chronic interstitial pancreatitis.

The following case illustrates all the main features of a typical pancreatic pseudo-cyst:

Case.—The patient, a girl, æt. 30 years, unmarried, by occupation a parlourmaid, came to my dispensary on October 31st, complaining of a pain in her stomach and of vomiting.

She stated that she had been perfectly well up to a month previously, when, while riding her bicycle

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near Clondalkin, she ran into a cart and was struck in the upper part of the abdomen by the shaft. She was thrown to the ground, but was able to get up and to ride into Dublin, where she went to a hospital for examination, but was not detained as a patient. She then went home, and feeling sick, went to bed, and remained there for five days. During this period she felt a lump in her stomach, and was sick on several occasions, but was able, nevertheless, after the rest, to resume her work as parlourmaid. A few days before coming to see me the swelling in the stomach got much worse, and she again began to suffer from vomiting and nausea. On examination in the dispensary a large rounded swelling was found in the abdomen. A provisional diagnosis of pancreatic cyst was made, and the patient was admitted to hospital.

A complete examination was made that evening. The patient was noticed to be considerably emaciated, and she herself stated that she had lost a good deal of flesh since the injury. The thoracic organs were normal. The upper zone of the abdomen was found occupied by a large, rounded swelling, which projected a little more prominently on the right than on the left side. This swelling was dull on percussion, and gave an obscure sensation of fluctuation. It did not move on palpation or with respiration. In the dorsal position it presented a very distinct pulsation, which was of somewhat expansile character; but this pulsation entirely disappeared in the upright and in the knee-chest positions. The circumference of the abdomen at the umbilicus was 31 in. As far as could be ascertained, the swelling was situated between the liver and the stomach, as no resonant note could be obtained between it and the liver margin. A very distinct splashing sound was detected with ease in the left iliac fossa, and was regarded as being due to extreme displacement and dilatation of the stomach. This view was subsequently confirmed at operation. Colon resonance was present along the right margin of the swelling.

Amongst the other features of the case it may be mentioned that the motions were normal in colour and consistence; the urine contained a faint trace of albumen and a few granular casts. A blood examination showed reduction of the red cells to 3,200,000 with 65 per cent. of Hb.; while the white cells were increased to 20,000 per c.mm. A differential count showed 82 per cent. neutrophiles, 14 per cent. lymphocytes, 3 per cent. hyaline cells and 1 per cent. basophiles. No eosinophile cells were noted in a count of 300 cells. The temperature was slightly raised, reaching on one occasion 101° F.

On the day subsequent to the patient's admission Mr. Stoney was asked to see her, and as he agreed in the diagnosis of pancreatic pseudo-cyst, it was decided to operate on the following day. On each of the two days in hospital prior to operation, and also on the morning of the operation, a large quantity of bilious fluid was vomited.

On November 4th, under ether anæsthesia, the abdomen was opened in the middle line above the umbilicus. A large tumour at once presented in the wound. It was tense, with a comparatively thin wall, and evidently contained fluid. It apparently filled practically the whole abdomen. It was covered by the anterior margin of the liver above, and until the incision had been enlarged below the umbilicus the stomach was not visible. It was then found that the stomach was enormously dilated and surrounded the tumour on the left and below. Gauze wipes were packed under the edges of the incision between the abdominal wall and the tumour. An aspirating needle was inserted, and about 10 oz. of a yellowish, somewhat turbid fluid evacuated. This reduced the tension in the cyst and allowed the wall to be grasped in clip forceps and drawn into the wound. An incision was then made through the cyst wall, and about two pints more of fluid were evacuated. The last of the fluid was blood-stained from contamination with blood from the incision. The posterior part of the interior of the cyst was irregular, and a mass was felt which was thought to be the body of the pancreas. There was some semi-gelatinous material in the depths of the cyst adherent to the wall. Some of this was removed for examination. The cyst-wall was palpated with one finger inside and another outside, but there was no sign of tumour or other cyst. A stomach tube was passed, and over two pints of green, bile-stained fluid were removed. Before this the lesser curvature of the stomach was on a level with the symphysis, and the greater curvature almost on the floor of the pelvis. The cut edges of the cyst were stitched to the peritoneum in the upper part of the abdominal wound in order to draw the stomach somewhat upward, and the abdominal wound was closed with catgut and Michel's clips. A few silkworm gut through-and-through sutures were also passed. A large drainage-tube was placed in the cyst and voluminous dressings were applied. The patient was returned to bed, the foot of which was raised with the idea of assisting the return of the stomach to its normal position. The importance of this was shown by the fact that there was no vomiting during that day or the next, but on the Monday,

as the patient said she did not like the position, the bed was lowered to the level. That afternoon the patient complained of nausea, and vomited during the night. The foot of the bed was therefore elevated the following day, when the nausea and vomiting ceased and did not return. On the second day the dressings were changed, as there was a considerable oozing of blood-stained fluid. On the third day the patient was dressed there was very little discharge, the skin was covered with zinc-oxide ointment, over which boric powder was shaken. On the fifth day the Michel's clips were taken out; there had been some discharge of fluid, and about 2 drm. of a thin, yellow, slightly blood-stained fluid were drawn out by a syringe. From this on the case was dressed daily, as there was a moderate discharge of thin watery fluid. The silkworm-gut sutures were removed on the eighth day, and the drainage-tube on the tenth day, a small gauze drain being substituted for it. By the end of a fortnight the amount of fluid escaping daily was greatly reduced, and only slight excoriation of the skin had occurred immediately around the opening. Calcium hydrate was then substituted for the boric powder, and after four days' application the excoriation had disappeared. Five weeks after the operation the patient was practically completely recovered, but a small sinus persisted, from which about a drachm of fluid escaped daily.

The fluid removed from the cyst was in quantity over 45 oz. It was alkaline in reaction, with a specific gravity of 1012. The first portion removed was of a yellowish, turbid character; the latter portion was stained with blood, and contained a considerable quantity of mucinous material, somewhat similar to what is found in an ovarian cyst. Microscopical examination revealed the presence of numerous polynuclear cells loaded with granules of fat, and of red blood-corpuscles.

Dr. Caldwell, of the Physiological Laboratory, Trinity College, Dublin, kindly examined the fluid for the presence of ferments, and reported that it contained large quantities of alkali-albumen, and also of proteoses and peptones in solution. Fibrin was rapidly digested, proving the presence of a proteolytic ferment, presumably trypsin; active starch and fat-splitting ferments were also found, the fat-splitting ferment being especially active.

The report of this case is, perhaps, mainly interesting from the way in which it illustrates the striking features of the group of cases to which it belongs. The analysis of the fluid proves without doubt its origin from the pancreas, while the anatomical conditions found at operation were consistent only with the assumption that the boundaries of the cyst consisted of the walls of the lesser sac of peritoneum. The extreme dilatation and displacement of the stomach is also a feature of note.

July 7th, 1913.

OLD AGE: ITS CARE AND TREATMENT IN HEALTH AND DISEASE. By ROBERT SAUNDBY, M.D., Edin.; Hon. LL.D. St. Andrews and McGill; Hon. M.Sc.Birm.: F.R.C.P.Lond.; Hon. F.R.C.P.Ireland; Professor of Medicine in the University of Birmingham; Consulting Physician to the General Hospital, Birmingham, to the Birmingham and Midland Counties Eye Hospital, and to the West Bromwich Hospital; ex-President, formerly President of Council, of the British Medical Association; Emeritus Senior President of the Royal Medical Society. Pp. vii+312. London: Edward Arnold, 1913. Price 7*s.* 6*d.* net.

IN this book there are brought together for the first time in recent years the various contributions that have been made in modern times to the care of old age in health and disease, including also the results of the author's forty years' experience. His object is first to show how a healthy old age may be reached; and secondly, to describe the causation, prevention and treatment of the diseases to which the aged are liable.

In the first two chapters are discussed the duration of life and normal old age. The next seven are devoted to disease as it affects the various systems—nervous, circulatory, respiratory, digestive, and genito-urinary. Two chapters are devoted to diathetic and general infectious diseases, and the last deals with regimen, dietary, and general treatment. There are six appendices, the first five of which contain useful prescriptions, dietary tables, recipes, and food analyses. The last appendix has been contributed by Dr. Mary Coghill-Hawkes, Principal of the Swedish Institute, and Medical Officer in Charge of Massage and Medical Gymnastics at St. Mary's and the Royal Free Hospitals. It contains a table of physical exercises for elderly people. There is a comprehensive bibliography, and an exhaustive index.

This book can be confidently recommended, alike for its valuable subject-matter and its interesting style. The author ably replies to the question from Ecclesiastes, "Why shouldest thou die before thy time?" with which he heads his preface, and he shows how by a proper knowledge of, and attention to, the principles of health, old age may be prolonged, and the diseases to which it is prone may be avoided or appropriately treated.

A Text-book of Midwifery. By R. W. Johnston, F.R.C.S., M.R.C.P.E., Assistant to Professor of Midwifery, Edinburgh University. Cr. 8vo, pp. 480, 264 illustrations. London: Adam & Charles Black. 10*s.* 6*d.* net.

This work forms an introduction to the "Edinburgh Medical Series," edited by Dr. John Comrie, and does it well. Whereas books on the subject are generally so large and cumbersome, in this one the subject has been treated most fully, and yet kept within the limits of an easily handled and "light" book. The early development of the embryo, the embedding of the ovum, and the development of the placenta, subjects which puzzle the student so often in their presentation, are here treated in a manner obvious to the veriest tyro.

Dr. Johnston, according to current fashion, treats the management of labour as a major surgical operation. Many suggestions, perfectly good in themselves, are crowded out by time and circumstance in the life of the average practitioner. In a future edition, which will not be long delayed, a condensed and simpler technique, suitable for the ordinary household and the routine practitioner, might with advantage be introduced. As might be expected from one who has worked for some time at eclampsia, the chapter on toxæmias of pregnancy is most suggestive. The author does not extol the use of scopolamine in general practice, because the practitioner *must* stay beside his patient. This largely defeats one of the hoped-for advantages in giving this drug, so that its use seems likely to be confined to hospital practice. In the work dealing with diseases associated with pregnancy are many helpful points to the general practitioner. Was it not Head who observed that glycosuria at that time was always associated with a female child?

The book is illustrated generously, and those bearing on post-partum hæmorrhage, extra-uterine gestation and performance of version will aid and guide the reader far more than bald statements of direction. A brief summary of infant feeding will refresh the mind of the busy practitioner, who has pertinent questions and suggestions made to him at that period. An extensive and reliable index completes a work for which we have nothing but praise.

Lectures on Diseases of Children. By Robert Hutchison, M.D., F.R.C.P., Physician to the London Hospital, etc. Third edition. Cr. 8vo, pp. 404. London: Edward Arnold.

A third edition of this work testifies to its usefulness in meeting a demand for a work which shall attractively and fully comprehend the various and apparently increasing diseases of children. These lectures, now bound together, were published serially in the Clinical Journal, a medium whereby many notable publications have reached the practitioner. This is an age of dedications, and this work is ascribed to Dr. John Thomson, colloquially known as the baby's "Sherlock Holmes." There is an illuminating chapter on infants' feeding, perhaps (next to training) the most tiresome and trying subject to the family doctor, in which the points of diagnosis and the corresponding methods of treatment are admirably blended. The author's style is attractive, and one on referring to the work for any particular object is temptingly encouraged to read on and on. There are sixty-nine illustrations and thirteen diagrams in the book, which should find its niche in every practitioner's bookcase.

Vicious Circles in Disease. By Jamieson B. Hurry, M.A., M.D.Cantab., ex-President Reading Pathological Society. Second and enlarged edition. Crown 8vo, 280 pp. London: J. & A. Churchill.

This book represents the first attempt to deal systematically with vicious circles in disease. These are classified into seven groups or natural orders. The subject is interesting, and the writer has a readable and easy style, which is used constantly to introduce extracts from authorities. In Chapter II, on "Circles associated with the Nervous System," there are twenty-four quotations; in Chapter III, on "Circles associated with the Cardio-Vascular System" there are thirty, and so on. Most of these could be dispensed with or placed at the end of the book, where they could be found for reference if desired. There is much in the book which will repay study.

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THE VALUE OF PAIN AS A SYMPTOM OF RENAL DISEASE.*

By J. SWIFT JOLY, F.R.C.S.,
Assistant Surgeon to St. Peter's Hospital for Stone.

IF one believed the illustrations that are to be found in the advertisements of certain proprietary remedies one would come to the conclusion that all forms of backache, and indeed that all pains felt anywhere between the base of the sacrum and the scapulæ, are due to renal trouble. This is obviously not the case; and as a matter of fact in the illustrations I have seen—illustrations where every picture tells its story—I do not remember ever seeing one in which the sufferer is depicted with his or her hand placed on the spot where renal pain is commonly felt.

Before discussing the diagnostic value of pain in relation to kidney disease it is necessary to have a clear and definite conception of the situations where renal pain is felt, and also of the causes by which it is produced.

Pain due to visceral disease (1) is divided into two categories: (*a*) local pain; (*b*) visceral referred pain. Local pain is felt directly over the part affected; it is due to irritation, generally microbic in origin, of the organ in question, and is increased by deep pressure. The best examples of such pain are to be found in cases of inflammation of the serous membranes—the pleura, pericardium, peritoneum, etc. However, it can be easily demonstrated in the case of the bladder, but in the case of the renal pelvis it is liable to be confused with the referred pain. The reason of this will be explained later.

Visceral referred pain is due to stimuli that are conducted up the sensory branches of the sympathetic to the spinal chord. There they produce

* Lecture delivered at the Medical Graduates' College and Polyclinic.

some disturbance which profoundly alters the nerve impulses reaching that segment through the nerves of ordinary sensation. What the nature of this disturbance is is at present unknown, but its effect is to produce a sensation of pain accompanied by superficial tenderness somewhere in the distribution of this particular nerve. If the disturbance is very great this pain may be felt over the whole of the sensory area of that nerve; in certain cases it may even cross to the other side of the body and thus produce a girdle pain. In this way, disease occurring in one of a pair of organs may give rise to pain on the opposite side of the body. However, this is uncommon; sometimes the pain takes the form of a half girdle and extends from the spine to the middle line in front, following the course of one of the segmental nerves on the side affected. Usually, however, certain definite spots seem to be picked out to which these painful sensations are referred. For each nerve they are two in number, one corresponding to the posterior, and the other to the anterior primary division of the nerve. Hence in visceral disease we get two chains of painful areas; one of these runs down the back parallel to and a short distance from the spinal column; the other has an almost corresponding position on the front of the trunk.

Now as certain organs migrate during the process of development from the position where their first rudiments are formed, it follows that the areas in which this referred pain is felt may be far removed from the spot where the mature organ is situated. The testicle forms an excellent example of this. It is in its origin an abdominal organ, and its first rudiments are laid down in close relationship to the kidney. During development it migrates downwards into the scrotum. Now, if this organ becomes inflamed, the local pain is felt in the scrotum, while the referred pain is situated in the areas of the lower dorsal nerves, especially the tenth, eleventh, and the twelfth, and the patient complains of pain in the loin and abdomen.

If, however, an organ does not migrate in this manner, and if its position when fully developed corresponds segmentally to that which its first rudiments occupied, one would expect that the areas of referred pain are anatomically in close relationship with it. Now this is exactly what we find in the case of the kidney. The kidney is more closely related to the tenth dorsal segment than to any other; the result is that referred renal pain is felt most in that area. The posterior pain area of this segment is situated immediately below the last rib, in the angle between it and the erector spinæ group of muscles, and the anterior area lies on the linea semilunaris at a level slightly above that of the umbilicus. But these are the very spots where deep pressure gives rise to renal tenderness and where local renal pain is felt. Therefore in the same region one finds superficial and deep tenderness, and direct and referred pain of renal origin. This is the reason why such confusion exists between the direct and referred pain of renal disease.

First, let us take up the question of visceral referred pain as it applies to the kidney and ureter. The great factor that seems to cause this pain is an increase of tension within the organ. Thus, blockage of the ureter by a stone increases the tension within the renal pelvis and gives rise to renal colic. Similarly the uterine contractions during childbirth enormously increase the intra-uterine tension and give rise to the characteristic pain in the back. Biliary colic may be explained in the same way.

Hence any condition that tends to increase the pressure of the fluid within the renal pelvis also tends to produce pain of the referred type; and, again, the more rapidly this pressure is increased the more violent is the pain. This statement, the truth of which is obvious to anyone who has watched the symptoms produced by the various types of renal disease and compared them with the findings of the operating theatre or post-mortem room, can be proved experimentally. If a catheter is passed up the ureter and if water be injected into the renal pelvis, any increase of pressure is accompanied by pain which resembles renal colic in every respect. The moment the pressure falls the pain ceases; when it is again raised the pain returns. This method has been used, especially in America, to determine whether obscure pain in the loin is due to a renal or to an extra-renal condition.

If one considers for a moment the kidney and ureter, one sees how easily the pressure may be raised sufficiently to cause pain. The capsule of the kidney and the layer of connective tissue that surrounds the pelvis and ureter, forming a sheath in which they lie, effectually prevent either of them

dilating before any sudden increase of the urinary pressure. Owing to the wide calibre of the renal artery and the fact that it springs directly from the abdominal aorta, the blood-pressure in it is very high. This pressure is transmitted almost directly on to the tuft of capillaries in the glomeruli, so that the excretion of urine—or at least of the watery parts of it—is carried on at a very high pressure. Taking into account the fact that water is practically incompressible, and that the ureter and pelvis do not dilate easily, one sees how any blockage of the ureter must cause an enormous increase of pressure within the renal pelvis, and that this increase of pressure is quite sufficient to give rise to the severest colic.

I stated a few minutes ago that the kidney was most intimately connected with the tenth dorsal nerve segment. It is also connected with the eleventh and the twelfth. Now, if one plots out the pain areas of these nerves one finds that the posterior areas are situated in the loin at a lower level than that of the tenth, while the anterior pain area of the eleventh is just in front of the anterior superior spine of the ilium, and that of the twelfth in the groin immediately above Poupart's ligament. Thus you see that the typical spread of a renal colic is mapped out by these painful areas. Again, tenderness of the testicle is sometimes noticed during an attack of renal colic. This is due to sympathy, both organs being supplied from the same level—the tenth dorsal.

Turning from this more or less theoretical discussion let us take up the practical side of the question. The points we have just considered will serve as a map to guide us through difficult and perplexing country.

The best known example of pain due to sudden increase of the renal pressure is a renal colic. This must not, however, be considered as pathognomonic of stone; it can be caused by the passage of blood-clots, fragments of new growths, masses of caseous material, etc., down the ureter, and even by a sudden blockage of the ureter from kinking. However, its most usual cause is stone. A stone anywhere in the upper urinary tract may cause renal colic; for example, a stone in the renal pelvis which is too large to pass down the ureter may suddenly block its upper end and cause colic; or a stone may remain fixed or nearly so in any part of the ureter and give rise to repeated attacks of pain; while lastly, we have the classic example of a stone passing down the ureter, this passage being accompanied by violent colic.

A renal colic due to stone is said to be complete or incomplete. If it is complete it causes the expulsion of the stone into the bladder; in an incomplete one this happy termination does not occur. However, several incomplete attacks may culminate in a complete one and the stone be passed.

It must not be assumed that the passage of all stones down the ureter causes pain. If a stone is small, smooth and round it may be passed painlessly. Many patients pass stones frequently yet they never experience pain; the pressure is never raised sufficiently to give rise to colic. Such patients pass uric acid stones as a rule; it is very rare for an oxalate stone to be passed painlessly. Oxalate stones are rough and crystalline, and the sharp edges of the crystals lacerate the epithelial lining of the ureter. This produces both œdema of the epithelium and spasm of the muscular coat of the ureter, with a result that the canal is completely blocked, the pressure in it rises, and an attack of colic ensues.

A typical renal colic generally has a sudden onset. It may come on without any cause that can be assigned for it, or it may follow exercise, any sudden movement or jolting. Thus walking, horse riding, or riding in any vehicle along a rough road, or a sudden jar such as is caused by a fall or by suddenly stepping off a curb-stone, may bring it on. Occasionally it is preceded by hæmaturia. The pain is very intense, and as a rule rises at once to a maximum. It is sharp and stabbing and is usually described as being like a knife. It commences in the back, in the angle between the erector spinæ muscles and the last rib, and then spreads round to the front to a point slightly below the tip of the tenth rib. This distribution is often described by patients as "the pain going through them." It next spreads downwards in front of the anterior superior spine of the ilium and along Poupart's ligament into the scrotum and down the inner aspect of the thigh. The testicle is usually retracted, and the abdominal muscles of the affected side are held rigid. Nausea and vomiting are present. The urine is scanty, tinged with blood, and passed frequently and imperatively. The picture of a man in the prime of life rolling

about in agony, and unable to find any position that will give him relief, is so well known that it hardly merits description.

The duration of an attack is very variable. It may last only a couple of hours, or the pain may be present almost without intermission for several weeks. Usually the shorter attacks are the more severe. If the attack is complete, and the stone is passed into the bladder, the termination is sudden and abrupt. The patient feels as if something had suddenly given way within him, and the pain ceases as if by magic. If it is incomplete the pain dies away slowly, and after a longer or shorter interval is again renewed.

Can one diagnose the position of the stone from the position of the pain? Generally this is impossible, but if the pain is only moderately severe a rough guess can sometimes be made as to its whereabouts. The reason of this is that in the extraordinary severe bouts of pain there is a tendency for it to be referred over a wider area than would be the case if the attack was less severe. Now renal pain is referred first to the tenth dorsal nerve area; if it is severe it overflows this area, and the eleventh and twelfth areas become involved. So that a stone which remains in the renal pelvis but temporarily blocks the upper end of the ureter may cause pain extending from the loin to the testicle. Pain with a similar distribution will be caused by a stone passing rapidly down the ureter. What happens if a stone passes very slowly down the ureter? In this case the patient has many bouts of renal colic extending perhaps over a period of several months. Furthermore the site of the pain gradually changes; at first it is felt chiefly in the back, some time later on in the side, and the last few bouts may give rise to pain almost entirely confined to the groin. As the position of the pain changes so does that of the stone. As a result of numerous obervations I am inclined to think that if the stone is in the renal pelvis or in the upper third of the ureter the pain is felt chiefly in the back. If it is in the middle third the pain is referred to the side just above the crest of the ilium, while pain in the groin points to the stone being in the lower third of the ureter. If a stone is impacted at the vesical orifice of the ureter the pain is referred to the vesical areas (tip of penis, etc.).

An attack of renal colic is accompanied by oliguria, the total amount of urine excreted during the attack being less than normal. The stone dams back the urine of the affected side, but this is often not enough to account for the diminution in the amount passed. Therefore the other kidney must excrete less than usual during the attack. If this "other" kidney is diseased—and in most calculus cases it is diseased—it may cease excreting during the paroxysm of pain. This gives rise to one form of calculous anuria—where the pain of a unilateral stone is sufficient to cause reflex inhibition of the excretory function of the other kidney. A patient recently under my care at St. Peter's Hospital illustrates this point. He was admitted complaining of pain in the right side. An X-ray examination revealed a large branching shadow in the right renal region, and a small round one low down in the line of the ureter of the same side. I cut down on the kidney and removed a large friable phosphatic mass that had moulded itself to the renal pelvis and calices. His condition did not warrant an attempt to remove the ureteric stone at the same sitting. He rapidly recovered from the operation, and left the hospital with his wound firmly healed. Two days after his discharge he returned with an intense right-sided renal colic, which lasted forty-eight hours, and ended with the expulsion of the calculus that was in his ureter. Now during this attack, although most careful and repeated examinations failed to reveal any evidence of stone on the left side, the patient was in a state of complete anuria. As soon as the pain passed off the kidneys resumed their functions, and urine was seen by means of the cystoscope to come from the left ureter.

An attack of renal colic is usually followed by a more or less marked polyuria, which passes off in a day or two. It is due to increased activity of the kidneys after the depressing effects of the pain has passed away. Both kidneys take their share in this increased elimination, but as the kidney of the unaffected side is less depressed by the colic than the other, the bulk of the urine is secreted by it. The same thing holds good in cases of intermittent hydronephrosis. A kink of the ureter or some narrowing of its lumen is always present in these cases. Now, if some sudden movement increases this kink, or if from any cause the urine cannot pass through the narrowed ureter, the pressure in the renal pelvis is suddenly raised, and

a sharp attack of pain ensues. This is accompanied by oliguria. It is usually stated that after a time "the hydronephrosis empties itself, the pain ceases, the tumour disappears, and a polyuria sets in." This description is hardly true. The hydronephrosis does not empty itself, and I doubt very much if a hydronephrosis ever empties itself. I have never seen a hydronephrotic kidney on the operation table that was empty. What happens is that a small quantity of urine leaks out, the pressure falls, and the hydronephrosis, instead of being tense, becomes flaccid. Now it is extremely difficult to feel a flaccid cyst that is placed as deeply as a kidney, therefore the saying "the hydronephrosis empties itself and the tumour disappears" should be altered to "the hydronephrosis becomes flaccid and therefore cannot be felt as a tumour." But you will ask, how can one account for the polyuria? This phenomenon is evident, and cannot be merely an error of observation. The polyuria certainly is present, but it does not bear any definite relationship to the size of the hydronephrosis, it is proportional to the intensity of the pain and not to the size of the tumour. In reality it is due to increased activity of the other kidney, and comes almost entirely from the sound side.

Up to this I have spoken only of the acute attacks of paroxysmal pain we call renal colic, but I do not for a moment intend to imply that this is the only type of pain associated with renal lithiasis. As a matter of fact the majority of patients who have a stone in the kidney, of such a size that it can only be removed by surgical interference, never have a typical renal colic. The most usual type of pain is a deep-seated stabbing pain that is brought on by exercise or jolting and relieved by rest. This pain remains localised to the renal region, it is felt posteriorly in the angle between the erector spinæ muscles and the last rib, and does not tend to spread round to the front or down into the groin. It is due to mechanical irritation of the renal pelvis by the stone. Probably this is increased by a spasmodic contraction of the muscular tissue of the renal pelvis, which in turn causes the stone to irritate the epithelium still more. In this way a vicious circle is set up. The fact that the pain comes on so acutely and suddenly after a brusque movement or shock is in favour of the hypothesis. Now the presence of spasm has the same effect on the nerve-endings as an increase of the hydrostatic pressure within the renal pelvis, therefore this type of pain must be considered as a combination of the local pain and the visceral referred pain. It is exactly analogous to the pain of a vesical calculus. A stone in the bladder causes pain at the end of micturition, which is felt near the tip of the penis. This is a referred pain, and is caused by the spasmodic contraction of the bladder on the calculus. When the bladder is full the urine acts as a water cushion between the vesical wall and the stone, and thus little or no pain is felt.

The objection will be raised that referred pain is not accompanied by deep tenderness, while in every case I have mentioned up to this—renal colic, hydronephrosis, pelvic stone, etc.—deep tenderness is present. The pain is always increased by pressure or palpation. The answer is that the act of palpation must in all cases increase the tension which causes the pain. In renal colic or hydronephrosis this is obvious; fluid being almost incompressible, any palpation of the kidney when in a state of retention must increase the tension within it, while in the case of a pelvic stone palpation of the kidney presses the epithelium of the renal pelvis against the rough stone and causes a reflex spasm. Thus the apparent anomaly of referred pain co-existing with deep tenderness is simply due to the fact that the painful areas are situated at the very spots where the finger-tips are placed when endeavouring to palpate the kidney.

It follows, too, from these considerations that a movable stone *ceteris paribus* is more likely to produce pain than a fixed one. The former is more likely to excoriate the renal pelvis or to block the upper end of the ureter on any sudden movement. A fixed stone does not necessarily mean one that prevents the outflow of urine from the kidney or from one part of it. It is usually only the crystalline points that are caught and fixed in the lining epithelium of the renal pelvis, and between these points there is plenty of room for the urine to trickle away. If, however, by its growth it does come to block the urinary outflow, this blockage comes on so slowly that it does not cause pain. Thus on rare occasions it happens that a kidney may be slowly and silently destroyed by a stone that has given rise to little or no symptoms.

Local Pain.

Now let us turn to those diseases of the kidney in which the intra-renal tension is not increased. Here a totally different train of symptoms is set up. In the conditions I have just described pain is the predominant symptom, in those we must now consider pain is conspicuous by its absence. There can be no greater contrast in this respect than renal stone and renal tuberculosis. Though both diseases, if left untreated and allowed to pursue their natural course, end in the destruction of the kidney, yet the one only achieves this end after years of almost constant pain, while the other may reach a like result before one suspects that the organ is even attacked. In renal tuberculosis the symptoms are practically altogether due to irritation or ulceration of the bladder, which is caused by the constant passage of highly infective urine through it. Of late years our ideas of urinary tuberculosis have been completely revolutionised. Formerly it was thought that the bladder was the organ most affected, and the only form of renal tuberculosis diagnosed clinically was the comparatively rare type where the kidney was markedly enlarged and formed a tuberculous pyonephrosis. But this type is not a pure tuberculosis of the kidney, it is a tuberculosis plus retention, or tuberculosis plus a secondary infection. Now we know that in the majority of cases the affected kidney is not enlarged, is rarely palpable, is usually painless, but may give rise to slight tenderness on palpation, and that the diagnosis can be made only by catheterisation of the ureters and examination of the urines of the two kidneys thus separated. The cystoscope gives us practically all the information about these cases on which our diagnosis is based; without it one could only give a vague guess as to the condition of the kidneys. Usually one sees one side of the bladder and one ureteric orifice eroded and ulcerated while the other is healthy. Although much can be inferred from the appearance of the ureteric orifices in these cases—a subject which I have not time to discuss here—catheterisation of the ureters is still necessary to prove that the other kidney is healthy and is able to carry out the needs of the body.

Occasionally, however, we find patients who complain of a definite renal ache in tuberculous disease of the kidneys, and if it is present it is most valuable, as it gives us the only clue (apart from cystoscopy) as to which side is affected. Generally it is very slight; often patients think it is too trivial to speak of until they are questioned about it. It is constant, confined to the renal area, unaffected by exercise, but is accompanied by tenderness on palpation. Thus, you see, it differs markedly from the pain of stone. I believe that this ache is due to scalding of the eroded renal pelvis by the irritating urine that constantly flows over it. In rare cases one may have a renal colic produced by the passage of a mass of caseous material down the ureter. This is a mere accident in the course of the disease, and in no way affects the statement that pain is not a prominent symptom of renal tuberculosis.

The pain of pyelitis, no matter what organism it may be due to, is another example of local pain. However, I thought it better to put tuberculosis first in this discussion, as, on account of the complete absence of retention, it forms the purest example of local pain I know of. In ordinary pyelitis the inflammation is more acute, and the mucous membrane of the pelvis and ureter become swollen and œdematous. This makes it exceedingly difficult to prove that the outflow of the urine is not hindered, and that there is no retention within the renal pelvis. Therefore, one is in doubt whether to place the pain of pyelitis in the category of local pain or in that of referred pain. Personally I think it is a local pain, especially as it resembles the pain of tuberculosis in every respect except that it is much more severe. One might say that it is an exaggerated reproduction of it, and as the inflammation is more acute in pyelitis than in tuberculosis, so is the pain. With this difference, the description of one will serve for the other.

Inflammation is not necessary for the production of a renal ache; it may be present in cases where the chemical constitution of the urine is markedly altered. The most common chemical change in the urine which produces this pain is that of increased alkalinity. This causes a precipitation of the phosphates, which is called phosphaturia. In most instances this term is a misnomer, as the total amount of phosphates in the urine is not increased, but they are only thrown out of solution by the alkalinity of the fluid. True phosphaturia —where there is a definite increase of the total amount of the phosphates excreted—is a compara-

tively rare condition. Be this as it may, this so-called phosphaturia often produces a definite renal ache, which disappears when the urine becomes acid again. I have had many of these cases submitted to an X-ray examination without finding any indication of calculus formation.

Lastly, posterior renal aching is a very important sign of renal failure in cases where there has been a long-continued back-pressure on the kidneys from stricture, enlarged prostate, or any other cause which prevents the outflow of the urine. Under these conditions the pressure is increased so gradually that the pain cannot be considered as due to it. It is met with both in septic and in aseptic cases, but in the former it occurs earlier than in the latter. In this type of case the kidneys are generally small, firmer than usual, and somewhat dilated. The cortex is very much thinned, and the microscope shows extensive fibrotic changes by which the secreting tissue is reduced to a minimum. This pain is a very serious symptom, as it indicates that the excretory function of the kidney is profoundly affected, and it is often the first warning of impending renal failure. It is a dull ache which is felt posteriorly in the angles between the erector spinæ muscles and the last ribs. Usually it is bilateral, and is unaffected by exercise, jolting or movement. In aseptic cases there is no tenderness on palpation. If, however, the case is septic, an ascending pyelonephritis is usually present, and this gives rise to tenderness on pressure. The pain generally increases with the onset of uræmia, but may disappear entirely if the cause of the obstruction is permanently removed. The causation of this type of renal ache presents a difficult problem. It does not fall under either of the headings I have mentioned, and, as far as I know, no really satisfactory explanation has been given to account for it.

Reference.

(1) 'Quain's Dictionary of Medicine,' article on "Pain in Visceral Disease," by Dr. H. Head. Anyone who is acquainted with this article will see how frequently and how extensively I have drawn from it.

July 14th, 1913.

SOME ACUTE ABDOMINAL PERILS.*

By J. W. LEECH, M.D., M.S., F.R.C.S.E.,
Hon. Assistant Surgeon, Royal Victoria Infirmary, Newcastle-on-Tyne.

Gentlemen,—On being asked to read a paper before the members of your Division on a subject of interest and practical value to practitioners, I selected abdominal perils. Time does not allow me to deal with the many conditions which the title embraces. Therefore I shall devote my remarks to the commoner perils.

I define an abdominal peril as an acute traumatic or infective disease occurring within the abdominal cavity, associated with urgent symptoms, among which pain, shock, muscular spasm and vomiting are outstanding. The great increase of our knowledge of abdominal lesions has tremendously increased the responsibility of the surgeon, and, needless to say, these responsibilities are keenly felt. The difficulties of the operation of the various abdominal perils are usually in direct proportion to the duration of the disease.

If an early diagnosis is made, not only is the general condition of the patient better for anæsthesia and operative manipulation, but the procedure itself is much simpler and safer, and the results infinitely better. Indeed they depend more upon early recognition than on operative technique. Upon this I can speak from a long experience of emergency work, and I would ask that all cases of abdominal pain should be regarded with suspicion; that we should cease to regard a pain as a touch of indigestion, an attack of colic and the like. It is expressive of a something wrong and demands investigation.

Pain, muscular spasm, rigidity and vomiting are perhaps the most positive signs of an acute abdominal lesion. As these are common to the perils upon which I shall speak, whether the initial lesion be a perforation or an obstruction, the fatal issue will in most cases be due to the same cause—peritonitis—though, of course, death may be due to shock in some cases of perforation of stomach, or from hæmorrhage following a ruptured ectopic gestation.

* Read before the Sunderland Division of the British Medical Association.

As the majority of cases manifesting perilous symptoms are due directly or indirectly to involvement of the peritoneum, I may observe that this membrane is highly sensitive, and that its sensibility varies in different regions. Lennander has shown that the parietal peritoneum is excessively sensitive, while the peritoneum of the stomach, anterior portion of liver, mesentery, great omentum, etc., is for the most part insensitive for all operative work. Moreover, according to Cushing, the peritoneal regions which lack feeling in health, lack it equally when inflamed.

The absorptive powers of the peritoneum are remarkable, varying like sensibility in different regions of the abdomen, according to the distribution of the lymphatics. It is important to bear this in mind in septic abdominal cases, as the position in which the patient is placed may prove the mending or ending of the case. The absorptive power is greatest in the upper abdomen—in the region of the diaphragm, where the vessels and stomata are numerous and large. This is therefore the most dangerous zone. In the middle abdomen the vessels are smaller and less numerous. In the pelvis they are few, and this is therefore the least dangerous zone.

Therefore, in an infective peritonitis, absorption of septic products will be rapid in the diaphragmatic area, less so in the middle area and less still in the pelvis. The practical lesson, therefore, is—that in peritonitis a patient is less likely to absorb toxins if placed in the sitting posture than in the recumbent. Moreover such posture would relieve respiratory and cardiac embarrassments. As opposed to the absorptive power, the peritoneum possesses a wonderful power of resistance, demonstrated by the outpouring of an exudate rich in phagocytes, directly the membrane is involved, thus tending to limit the area of infection on the one hand and of diluting existing toxins on the other, likewise inhibiting or destroying bacterial growth. It is to this virtue that an inflammation is prevented from spreading, the exudate surrounding the infected area becoming converted into plastic lymph, with the result that a localised abscess is formed, as is often seen in appendicitis. This protective reaction may completely clear the field of action and the patient recover without further disquieting signs. This salutary process does not follow all infections; the resistance may be overcome by the virulence of the infecting organisms, and furthermore, the anatomical area involved may be a factor operating against its influence. That the exudate is for good is universally recognised.

Peritonitis, in the sense in which I shall use it to-day, is of exclusively septic origin, and due to direct infection by contact or indirect infection from the presence of toxins invading the peritoneum through the circulation. It is not, therefore, a disease *per se*, but rather a formidable sequel to other abdominal lesions—in themselves less serious—but eventuating in peritonitis. Such are perforation of a gastric ulcer, perforation of an appendix, direct passage of organisms through a necrotic intestinal area in cases of obstruction and penetrating wounds of the abdomen.

As pain is perhaps the first warning to the patient of a serious internal mishap, though to the doctor not the most important or most trustworthy, we should endeavour to recognise its significance, its character and behaviour in acute abdominal lesions. Similarly should we investigate other signs of abdominal perils, such as rigidity, vomiting, etc. A timely recognition of their significance, with a due appreciation of their importance, may be of inestimable help in arriving at an early diagnosis, and thus enabling an appropriate treatment to be carried out. A disregard to such signals of danger as pain, vomiting and rigidity will lead us into inextricable difficulties, along thorny and rocky paths to an abyss of hopelessness, with no means of escape. To be confronted with the lurid picture of a disseminated peritonitis, the result of a delayed recognition of earlier symptoms, is a grave reproach to our professional skill.

An acute abdominal pain may be transient or persistent; it may be aggravated by the patient's fear; may be due to causes entirely outside the abdomen, though definitely focussed by the patient to a limited field. You are all aware, for instance, of the acutely painful abdomen of pneumonia, and the excruciating gastric crises of tabes. A general exclusive method of examination should, therefore, never be neglected in investigating the cause of an abdominal pain. Furthermore, diseases of abdominal organs frequently account for pain in regions far removed, such as shoulder-pains symptomatic of lesions of the liver, the tender spine of gastric ulcer, and the painful testicle of renal calculus.

Granted that the pain is of abdominal origin, what significance has it, and what lesson does it convey? It is common to all acute perils. We are to-day dealing with some of the commoner, such as obstruction, peritonitis, gall-stones, etc.

Intestinal Obstruction.

In intestinal obstruction from any cause—bands, Meckel's diverticulum, intussusception, etc.—pain is always present and manifests clinical characters of importance, almost pathognomonic, which at once give a clue to diagnosis, not necessarily in defining the exact nature of the obstruction, but suggesting that the pain is an obstruction pain, and thus distinguishing it from the pain of a strangulated ovarian or other tumour.

In acute obstruction the severity of the pain depends upon the suddenness of the strangulation and the amount of bowel involved. The pain is continuous in the earlier stages, but later, in an obstruction not yet complete, there is superadded a paroxysmal pain, colicky and violent, due to peristalsis of the gut and associated with great depression and vomiting. During the exacerbations the intensified pain becomes localised at the site of the obstruction, and at this point on palpation there is greatest tenderness.

You are all familiar with the changes in the gut following a strangulation—so clearly seen in an external strangulation—first the nipping, followed by venous obstruction, while the arteries continue their supply, consequent engorgement and œdema, with a greater or lesser exudation, later entire blood stasis, change of colour varying from deep red to claret or plum colour, loss of vitality of the walls, paresis, gangrene. These changes are productive of the train of symptoms completing the picture of obstruction. I shall briefly refer to some of these symptoms which are common to most acute lesions, and, therefore, are in no sense confined to cases primarily intestinal. These are meteorism, vomiting and rigidity.

Meteorism is essentially due to intestinal troubles, and is always present in a greater or lesser degree in all forms of obstruction. It is the rapidity of development and the degree of intensity which are immeasurably helpful. Whatever the cause, whether due to circulatory disturbances or obstruction in the gut, intestinal absorption is diminished, decomposition and fermentation rapidly takes place, and the coils become enormously distended with gas, diaphragmatic movements are interfered with, respiration becomes superficial and the heart's action embarrassed. Being essentially intestinal—leaving meteorism as incident to peritonitis out of the question for the moment—this somewhat rapid development, with accompanying great distension and general thoracic embarrassments, would at once suggest an intestinal obstruction of the volvulus type. In no type of obstruction is meteorism so sudden and severe as in volvulus of the sigmoid. Important, too, is the fact that meteorism is never diminished by vomiting.

From the point of view of treatment, it may appear to you perhaps immaterial where the obstruction is or how it is caused, and, indeed, with the exception of volvulus and intussusception, it is almost impossible—clinically—to distinguish the various types. In these two forms, the presence or absence of meteorism is typical; in the former it is a prominent feature, while in the latter it is almost or entirely absent. In obstruction by bands meteorism develops slowly. In this connection may I say that it would be a useful rule to regard intense meteorism as due to large gut involvement.

Vomiting is common to most acute lesions. It is important that the nature of the act and the character of the vomitus should be recognised in order that we may have a clue as to the lesion. It does not necessarily depend upon an obstructed bowel but may be entirely reflex, as in the vomiting which not infrequently ushers in an attack of acute appendicitis or the vomiting of an acute kidney lesion. It may be expulsive or projectile, or merely regurgitant, and this feature should be duly recognised and its significance interpreted. In acute obstruction, for instance, it is expulsive, occurring at varying intervals, suddenly, without nausea or effort, in large quantities and in great force, the vomitus at first being stomach contents, then bile-stained and later fæcal, the development of the latter depending upon the site of the obstruction whether high or low in the tract. In peritonitis, by way of contrast, the vomiting is persistent and accompanied by nausea and straining, while the vomitus is small in quantity and does not become fæcal, except at a late stage. Furthermore, it is usually of the regurgitant type.

It may be impossible in many cases to make an immediate diagnosis, but as acute abdominal cases

are perhaps the most perilous in surgery, the importance of making every effort to correctly interpret the picture presented cannot be exaggerated. Delay may spell disaster. How fatal may be the error, for instance, of mistaking in a child an intussusception for a general intestinal derangement due to a teething process. Such an error is not uncommon.

Whether the acute symptoms are due to an obstructed bowel, a strangulated ovarian pedicle, perforation of the stomach or duodenum, the fatal issue, if unrecognised and untreated, is due to the same cause, viz. peritonitis. A patient suffering from obstruction does not die because the flow of intestinal contents is arrested. The vitality of the bowel-wall has become so impaired that organisms pass through and infect the peritoneum and septic peritonitis supervenes, presenting a picture so lurid as never to be forgotten. The primary object, therefore, is to prevent peritonitis. This can only be done by early interpretation of the symptoms.

Intussusception.—Perhaps the one only variety of obstruction liable to be misinterpreted is intussusception. In general, obstruction cases are readily recognised by the history, the special character of the pain and vomit, the meteorism in its varying degrees, the constipation, and the shock, etc. In intussusception, however, the picture is not always clearly that of obstruction and the history is often misleading, occurring as it usually does in an infant, perhaps when teething, by the sudden onset of pain, vomiting, and it may be by the immediate passing of blood and mucus which the mother regards as an incident of dentition, the blood and mucus being looked upon in the light of a salutary diarrhœa. Little attention is paid to this, and the child apparently settles down again quietly and the doctor's services are not sought. In an hour or two the child cries out again with pain, a pain now superadded to the original one and due to intestinal contraction. The doctor is now sent for. The spasm passes off, though the vomiting continues at intervals, and on his arrival the child is apparently comfortable and easy. He does not see the infant during a paroxysm, perhaps does not note the bloody discharge from the anus, and accepts the mother's statement that the child is suffering from diarrhœa due to dentition. My experience is that a considerable number of these cases are overlooked in the early stage when operative treatment is so necessary and successful, while the later stages are well-nigh hopeless. The engorgement of the intussusception passes on to complete stasis, eventuating in gangrene.

I have remarked that meteorism is almost entirely absent in these cases, certainly in the early stage; the abdominal walls are flaccid, there being no rigidity. There is thus no obstacle to abdominal examination, which in all cases—no matter how small or how recent the intussusception—ought to reveal the presence of a tumour. This tumour will necessarily vary in size, shape, and position, according to—

(1) The variety of intussusception.
(2) To the amount of gut invaginated.
(3) The degree of engorgement.

Called, then, to a child, say, under twelve months of age, with a sudden history of pain and vomiting, suspect an intussusception. It is the commonest abdominal peril in infants. Your suspicion would be confirmed by examination—the flat abdomen, void of rigidity, because there is as yet no peritonitis, the presence of a tumour. Should the child have a paroxysmal attack of pain during your visit, its hands will probably be directed to the seat of the pain, and this will be the site of the tumour. This tumour has been aptly described as sausage-shaped, but this, of course, depends upon the extent of the invagination. The presence of blood and mucus will be further confirmation of the diagnosis. Later, of course, the intussusception may be felt *per rectum*, but I trust that the case has been diagnosed ere this is present, as it indicates great advancement of the lesion and a grave prognosis.

Granted an early diagnosis, what is the treatment? Emphatically—operation. There should be no attempt at palliation. The inflation treatment is obsolete, and the spontaneous reduction or passage of the gangrenous gut *per anum* most improbable. Operation is the only safe measure, and if undertaken within the first sixteen hours or so reduction is possible, and the results are good. Later, reduction will probably be impossible, owing to the swelling and engorgement, or possibly owing to the intimate matting together of the two tubes of intestine. In these cases even the slightest attempt at reduction may be fraught with danger, as the gut, possibly approaching the gangrenous stage, may rupture during the manipulative procedure. Resection or enterostomy are now the only alternatives.

The former can at once be put out of consideration as the shock would be beyond the child's resistance, and enterostomy, though saving the child is only the first of a series of operative procedures.

In the meantime, should operation not be undertaken, decomposition is taking place within the gut, the vitality of the walls becoming impaired, perhaps gangrene has developed, bacteria pass through the necrotic area infecting the peritoneum, and fatal septic peritonitis develops.

Volvulus is, perhaps, the only other obstruction which can be diagnosed as such. It should be suspected in the case of a man over forty, habitually constipated, being suddenly seized with pain, accompanied, it may be, with vomiting, which, however, is not urgent, and a great and rapid meteorism, which, as already stated, is more prominent than in any other type of obstruction. Volvulus is commonest in the sigmoid, owing to the constant stretching of its mesentery in constipated people, allowing a wide play of mobility.

Obstructions form a class of abdominal perils no less formidable than acute perforations, having many symptoms in common, but differing in this important respect: they present very definite signs and symptoms, which, if recognised and treated with expedition, will end well.

In perforations, on the other hand, the doctor sees the case only when a catastrophe has occurred, when the patient is in a condition of severe shock, from which he may die, or when there is disseminated peritoneal infection involving a grave prognosis.

Leaving gastro-intestinal perforations for later reference, I will briefly refer to one or two other abdominal perils commonly met with, such as:

(1) *Strangulated ovarian or myomatous pedicle.*
(2) *Ruptured ectopic gestation.*
(3) *Gall-stones and sequelæ.*
(4) *Ruptured pyosalpinx.*
(5) *Appendicitis.*

All give rise to peritonism in some form, and have symptoms in common with the class of cases already described.

Can we differentiate these cases? In the main history is important. In the first class—ovarian and uterine—it will probably be found that there has been a pre-existing tumour, though not infrequently a tumour exists without the patient being conscious of it. The symptoms are briefly:

(1) Pain—sudden and continuous.
(2) Shock.
(3) Vomiting.
(4) A tumour in the lower abdomen, presenting a dull note on percussion.

Comparing this condition with obstruction we find, by way of contrast, that the pain is continuous, the paroxysm due to peristalsis, so marked in obstruction, being absent; that the vomit is never fæcal; that the shock is always less marked; and that meteorism is absent. There is no true rigidity of the abdominal wall, this being present only as a result of peritonitis.

In twisted suppurating ovarian tumour the symptoms would be much intensified, the symptoms of inflammation being superadded to the above. The picture might then be of the gravest.

In ruptured ectopic gestation history is again important. A woman who has missed a period is suddenly seized with severe abdominal pain, vomiting and collapse. She immediately becomes blanched and the pulse is rapid and accelerating. There is tenderness, with some rigidity. Examination will reveal an enlarged uterus with blood escaping. The persistent pain unassociated with exacerbations, the rigidity from sudden efflux and involvement of the peritoneum, the rapid and increasing pulse with general blanching and the history of a missed period will generally suffice to differentiate from all other acute conditions. Immediate diagnosis and operation are imperative.

Gall-stones may create urgent symptoms, but the distribution and character of the pains are so definite as at once to rivet attention to the gall-bladder. This, with a possible history of jaundice and a tender, rigid gall-bladder area will confirm our suspicions. That gall-stones are the result of bacterial invasion is generally admitted. The subsequent changes in the gall-bladder, etc. (which, by the way, do not necessarily develop), are, therefore, inflammatory. While gall-stones are in the quiescent state the patient will generally complain of indigestion, sickness and pain about the stomach region, which may prove misleading. On investigation it is found that the pain and vomiting have no relation to the taking of food, but the fact that the vomiting apparently relieves the pain gives the patient the belief that he suffers from indigestion. Gall-stones produce a constant tension pain of a dull aching character,

more or less limited to the region of the gall-bladder, but it may be referred also to the subscapular region. This pain has to be distinguished from true hepatic colic, which is paroxysmal and may be excruciating, and, though accompanied by bilious vomiting, it has no associated constitutional disturbances. This excruciating pain is due to the escape of a stone or stones into the cystic or common duct, which undergoes spasm in the effort to expel the impacted body. The characteristics of the pain are the abruptness of onset and the suddenness of relief. It lasts only while the stone is in motion. The real dangers of gall-stones are due to inflammatory changes—suppurating cholecystitis, empyema of the gall-bladder, the spreading of infection from the viscus to the peritoneum by a perforation of its inflamed walls, directly setting up a septic peritonitis, or by a slower process of direct spread to the duodenum and pylorus, resulting in matting of the viscera incapable of separation, which in turn causes a mechanical obstruction.

Again, jaundice may result from inflammatory œdema of the common duct, or a stone may be dislodged and become impacted in the common duct, causing intermittent or persistent jaundice. It is true that gall-stones may remain quiescent in the gall-bladder for months or years; it is equally true that at any moment a disturbance may take place which hurls the patient with tragic suddenness from apparent health to grave peril. I urge that once gall-stones have been diagnosed, operation should be undertaken at the earliest moment, when all can be done thoroughly and safely, and the patient saved the perils which would in all probability follow delay.

Gastro-Intestinal Perforations.

Perhaps the most vulnerable of all acute conditions are those due to perforations. They are generally gastric, duodenal or appendicular. I need not dwell upon the signs and symptoms of gastric or duodenal ulcers, but would observe that in 90 per cent. of perforations there is a history, more or less typical of ulcer. I have recently had under my care two boys, æt. 11 and 15 years respectively, who suffered from perforations, without any premonitory symptoms, and without any previous digestive symptoms. A negative history, then, does not exclude ulcer.

Onset.—A sudden sharp abdominal pain, accompanied by shock which may be so marked as to cause sudden death, with marked rigidity of the abdominal wall, is an initial symptom in perforations. Immediately the facial expression changes to one of anxiety and great distress. Here we have at once a typical picture of peritonism, which markedly contrasts with the pictures previously alluded to. The pulse immediately becomes rapid and imperceptible while the respirations shallow. Vomiting may or may not be present. In subacute cases, of course, the symptoms are less marked.

On examining the abdomen the recti are rigid, and the abdomen feels like a board and does not move during respiration. At first the abdomen is retracted and later becomes distended with gas and fluid. Liver-dulness is absent as a rule, and if this is recognised at once is a most helpful clue as to what has happened.

The presence of free gas in the peritoneal cavity always suggests perforation of an air-containing viscus, and as the air immediately finds its way between the diaphragm and the upper surface of the liver the normal dulness is lost. This is a most valuable sign.

It must be borne in mind that in cases of great distension of intestinal coils following obstruction the liver may be pushed upwards and possibly rotated, with the result that the normal area of dulness is diminished. Distension from obstructive causes, however, with the exception of that from the volvulus type, is comparatively slow, and is not found for some time after the onset; while in acute perforations liver-dulness is at once obliterated.

Rigidity.—This is the most trustworthy warning of a grave danger. It is infinitely more valuable than pain, which may be referred from a distant lesion. Rigidity is only produced by acute inflammation of the serous membrane, the cause of which is a lesion demanding operation. Its usefulness cannot be over-stated. A kind of rigidity can readily be produced by passing a cold hand over the abdomen, or by deep pressure in examining the deeper parts, but this is not true rigidity of the abdominal wall itself, and consequently is not of grave import when compared with the true condition due to inflammation of the peritoneum—which may be local or general. True

rigidity at once appears on infection of the serous membrane, and is present in advance of the facial expression—described by Stanmore-Bishop as the hunted expression—so typical of septic peritonitis.

In cases of obstructive perils there is no immediate true rigidity, but there is an area of resistance and tenderness at the site involved, and true rigidity only asserts itself when from changes in the intestinal wall bacteria escape and infect the peritoneum. The same holds in twisted pedicle (ovarian), which in itself is not infective, but if by chance inflammatory infective changes take place within the twisted tumour, or if the tumour be septic before the strangulation occurs, then there is an immediate true rigidity.

In *appendicitis* (fulminating type) exactly the same process sets up perilous symptoms of peritonitis. The organisms are excessively virulent, the necrotic process is rapid, resistance is impaired, and perforation occurs ere the serous membrane has set up its defensive barrier, and general peritonitis results. Both a perforation of the appendix and a perforation of the stomach or duodenum culminate in peritonitis. But the stomach possibly discharges a large amount of contents suddenly, and the shock is great. On the other hand, the appendix, having no contents—in this sense—sets up a more rapid peritonitis from escape of its contained cocci; but the immediate shock is less. Thus rigidity is important, as indicating the nature of a sudden intra-peritoneal mishap. It is particularly helpful in relation to the time of its appearance after the onset of the lesion.

The foregoing remarks are applicable only to the spreading cases of septic peritonitis. But robust subjects with a good resistance present a less alarming picture, as adhesions are formed, which are protective in limiting the infection to a localised area. Thus in some cases of appendicitis a localised abscess forms after perforation; so also in subacute gastric or duodenal perforations, a localised abscess may result.

The diagnosis of appendicitis does not, as a rule, present any difficulties. The question is when to operate. It is true that some cases resolve and give no further trouble. Others resolve to relapse in a virulent form with development of a general peritonitis. In the majority of cases the sequence is somewhat as follows:

A catarrhal stage leads to ulceration, gangrene and perforation. In the meantime the peritoneum has been busily engaged throwing round the inflamed organ a protective barrier of lymph, and thus saving the general cavity from invasion. A localised abscess is the result. This in itself may not be for the moment a perilous state, but the abscess itself may eventually perforate and light up a general infection, when the picture becomes grave. A fulminating type is rarely primary. There is invariably a history of a previous attack which had resolved.

Now, in all cases of resolution the appendix is abnormal, that is, fibro-cicatricial tissue develops as a natural sequel to inflammation, which in turn constricts the lumen of the organ. The distal end of the appendix tends to dilate and becomes bulbous. If a further attack supervenes the dilated end rapidly undergoes a process of necrosis and perforation takes place. Operation should be undertaken in all but the mildest cases, that is, those which fail to show signs of abatement within twenty-four to thirty-six hours. After the lapse of this period, *should the pulse tend to increase in rapidity* with marked pain and rigidity over the appendix area, the case will be progressive, and at the best an abscess will follow. I do not think we are justified in allowing this to happen any more than to allow a general peritonitis to follow an intestinal obstruction.

If operation is undertaken at an early stage it is simple and safe. In the abscess stage it is fraught with risks and danger of damaging the viscera, of infecting the general cavity, and of septic phlebitis. The abdominal wall, owing to the necessity of drainage, is weakened and the convalescence is prolonged. Early operation, too, would probably avert the possibility of a fulminating type.

I have endeavoured to trace only a few abdominal perils from their onset to the development of a general septic peritonitis. Peritonitis from a ruptured appendix is much more virulent than that from a ruptured stomach, because it is due to virulent organisms, such as streptococcus, staphylococcus, and *Bacillus coli*. These are probably absent in the stomach. On the other hand, the shock is much greater in stomach perforations and may cause immediate death, while it is slight in appendicular rupture. So, too, a general infection from the gonococcus—rupture of a pyosalpinx—bears no comparison with

an appendicular infection. Moreover, the pus in a pyosalpinx is often sterile.

Any of the conditions described may result in general toxæmia (septic peritonitis), which will probably be fatal. There is no picture in surgery so awful, so hopeless and so forlorn. The face is drawn and pinched, the lips thin and blue, showing the coated teeth, the tongue dry, coated and tremulous, the eyes sunken, the expression anxious, the thirst intense. The patient lies in bed, helpless, distressed and restless to a degree. The abdominal walls are rigid and board-like. The knees are drawn up to relax the strain of the abdominal muscles. The pulse is small, rapid and wiry. This is of great prognostic value, indeed, the most reliable guide as to the condition of the patient, and once attaining 120, and still tending to increase and losing its volume, is of grave omen.

Follow the case further. The rigidity — so characteristic of peritonitis—now yields, and the patient will say he feels better. True! he is poisoned to death and does not know it. The pulse becomes quicker and quicker until imperceptible, the face becomes more livid owing to cardiac embarrassment and lack of oxygenation. The cyanosis, augmented by the toxæmia and weakened heart's action, increases. Cold sweating and a cold clammy skin complete the picture.

In the abdomen are many pitfalls for the unwary. The following are some of the conditions which mimic peritonitis. They should be ever present in your minds in dealing with acute diseases of the abdomen :

(1) *Acute nephritis.*—I have seen more than one abdomen opened for a supposed obstruction and nothing found—the lesion was renal in origin.

(2) *Acute pancreatitis.*

(3) *Toxic gastritis.*

(4) *Lead poisoning.*

(5) *Renal colic.*

(6) *Acute pulmonary diseases*, especially pneumonia—I have seen many cases admitted to the surgical wards as appendix cases.

What, then, are we to do when called to an abdominal peril? Give relief? Nothing of the kind. Make a diagnosis. By recognition of the significance of the symptoms this can be done and many disasters averted. Success in operative treatment depends almost solely upon the early recognition. Peritonitis is a serious menace; its death-rate is horrible. Recognise that acute symptoms means an acute lesion. The patient is in excruciating pain; he appeals to you for relief, and you are naturally anxious to give it. Perhaps your desire to do so over-rules your better judgment, and you give morphia. This is a grave and serious mistake. You have not made a diagnosis, and until this is made morphia should be surely withheld. Morphia will ease your patient; it will ease the pain; diminish shock; it will, too, inhibit peristalsis; will mask the symptoms, and will probably delude the patient, and possibly the doctor, into the belief that he is better and that all is going on well. Only later is the error seen when peritonitis develops—the cause of which you are ignorant. However, once the diagnosis has been made, morphia may be given with advantage pending the necessary arrangements for operation.

It would take too much time to detail the many methods of procedure which might be adopted in your investigations. Briefly, let me remind you of the onset, character and history of the pain, and the lesson it conveys. Of rigidity, whether present or absent: whether general or local. Of local rigidity, which is suggestive of the lesion present:

(A) *Fixed right rectus* of a pyloric or duodenal ulcer.

(B) *Fixed left rectus* of the cardiac end of stomach.

(C) *Local rigidity in right lower abdomen* over appendix area.

(D) *Localised* (*not true*) *rigidity* over an internal strangulation.

(E) *Rigidity in right upper abdomen* suggestive of gall-bladder lesions.

Bimanual examination.—Use to elicit the presence of any tumour in the various topographical areas.

Percussion.—Use as an aid to inspection and palpation, and particularly in determining the boundaries of liver, spleen, etc., and especially in recognising the presence or absence of liver dulness.

Vomiting.—Note its character when it occurs, whether explosive or regurgitant.

Vaginal examination.—Use to ascertain any abnormality in the fornices, such as pyosalpinx, ectopic pregnancy, ovarian tumours.

Rectal examination.—Use to ascertain presence of possible growth, accounting for acute intestinal symptoms.

Hernial sites. — Examination of these should never be neglected, as it is clear that a great error would be committed were an abdomen opened for an obstruction situated externally. Acute obstruction due to small knuckle of gut strangulated at the femoral ring in stout subjects may readily escape detection.

REFERENCES.

KEEN.—'Surgery.'
TREVES.—'Surgery.'
MOYNIHAN.—'Gall-stones.'
SARGEANT.—'Emergencies.'
FOWLER.—'Surgery.'
GOULD AND WARREN.—'Surgery.'

July 14th, 1913.

Labour in Paralysis Agitans.—A. Bonnet-Laborderie states that the advanced age at which paralysis agitans usually begins and the unfavourable conditions for conception render the following case a rarity.

A single woman, æt. 41 years, had suffered from paralysis agitans in a typical form for four and a half years. When seen for the first time by the writer in the eighth month of pregnancy she was unable to dress or feed herself or even to chew what was placed in her mouth. She could walk alone for only a few paces and could not get into bed without assistance. Her intelligence was intact, but her articulation was so much impaired that it was difficult to understand her. The pregnancy was perfectly normal and sickness and sympathetic disturbances were absent. At term spontaneous rupture of the membranes occurred after a few almost painless uterine contractions, which then ceased. Not till five days later did true labour pains begin. Dilatation was slow until the os attained the size of a 2-franc piece, when the patient's sufferings seemed to rouse her from her lethargic state and she began to make expulsive efforts of extraordinary vigour. A male child weighing 3350 gm. and measuring 51 cm. was born without assistance, and followed in twenty minutes by spontaneous expulsion of the placenta. She made a good recovery, but there was no secretion of milk.

The energetic contractions of the abdominal muscles during the second stage were remarkable. They resembled the efforts of a young and vigorous woman rather than those of an elderly and helpless patient, and showed that in spite of the muscular rigidity of the disease there is no loss of power if the patient will make the effort.—*Jour. des Sciences Méd. de Lille*, p. 361.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from page* 160.)

(227)

The Typical (?) Anglo-Indian.

In olden times as regards other subjects than malaria and fever, our notions of an Indian, or a European from India, were exceedingly erroneous; we thought that every man coming from India had a big liver, a big appetite, a big spleen, and a bad temper, and was jaundiced, bilious, and a gourmand. We know now that that description is erroneous; we have overcome that notion about the Anglo-Indian.

("On the Significance of Fever in Patients from the Tropics," by Sir Patrick Manson, K.C.M.G., M.D., F.R.C.P., F.R.S., 'Clin. Journ.,' vol. xxxiii, p. 305.)

(228)

The Value of the Microscope in the Diagnosis of Tropical Diseases.

The microscope is of more value in the diagnosis of a tropical disease than is the stethoscope in the diagnosis of lung disease, and were I given two instruments from which to choose for tropical disease I would discard the stethoscope and retain the microscope, so important is the microscope in tropical disease; it gives you precision, certainty, confidence. I advise any student before going out to the tropics to learn his blood examination well, to learn the use of his microscope in the diagnosis of blood disease, to learn to recognise a malaria parasite in all its phases. . . . It is not enough to have a microscope to be a microscopist. (*Ibid.*)

(229)

The Mental Factor in the Treatment of Disease.

Just one other point, gentlemen, and I have finished with the general management of heart troubles. Mental repose, we have stated, is a great element in sleep and in maintaining health; now a very great help to mental repose is an impression

of improvement made upon the patient himself, and this impression is almost invariably produced on a patient by permission to get up. His mental attitude is this: "My doctor is doing his best for me; he thought I was so ill that I must go to bed, he now thinks I am much better or he would not let me up, and, by Jove! I do feel better; I'll get up." Don't despise such homely feelings, gentlemen; they inspire hope, and where hope is there is a chance, and when hope departs from our patients I might say our functions as healers are practically useless, and only those of the parson and undertaker are of much avail. Remember this in all diseases, though mentioned only in connection with the heart.

("On some Diseases and their Management," by Dr. F. J. Smith, 'Clin. Journ.,' vol. xxxiii, p. 129.)

(230)

Unilateral Exophthalmos and Graves's Disease.

You will have noticed in our patient that the exophthalmos was bilaterally symmetrical. That is of very great importance. Occasionally an eye looks a little more prominent than its fellow, but I think you will be well advised if you hesitate a long time before you allow yourself to diagnose exophthalmic goitre if one eye is prominent and the other is not. Such cases are recorded but they are very rare; and nearly always when one eye is prominent and the other is not, the patient is really suffering from a tumour at the back of the orbit, which is pressing that one eye forward.

("Exophthalmic Goitre," by W. Hale White, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxi, p. 289.)

(231)

Human Tails.

But when you come to analyse and look critically into the reported cases of human beings born with tails you will find that they are not so, and that to call them tails is too elastic a use of the term. Most of them are either congenital tumours, which may be sacro-coccygeal tumours, or meningoceles or fatty tumours. There is an amusing story told relating to a famous old Scotch judge, Lord Monboddo. He believed that all individuals were born with tails, and that it was the duty of the midwife, when she attended a lying-in woman, to cut off the child's tail. On one occasion when Lady Monboddo was adding to the population the midwife heard something moving behind the curtains of the room, and on looking to see what it was she found him hiding and watching with the express intention of seeing her dock the child's tail.

("With Mr. Bland-Sutton at the Middlesex Hospital," 'Clin. Journ.,' vol. xxxiii, p. 97.)

(232)

The Importance of a Skilled Laryngoscopic Examination in cases of Prolonged Hoarseness.

The best way to find out why a patient is hoarse is to look into his larynx. But it is not everyone who can do that. But short of doing that one can sometimes "snigger at a surmise" as they say in America. The way to snigger at a surmise in connection with these things is to first of all realise whether there is an acute or chronic condition. That is why it is so important to obtain a careful history, not always trusting to the patient himself or herself. It should be a rule that no one should be allowed to be hoarse all the time for more than a month, or intermittently hoarse for a couple of months without getting a skilled laryngoscopic examination made. If the practitioner in whose hands the patient is does not feel justified in doing it, he should not incur the responsibility of treating the case; he should tell the patient simply that he wishes to have his larynx examined. He should not hesitate to have this done any more than he would hesitate to have the fundus oculi examined if the patient could not see out of one eye, because it is of vital importance at all ages of life.

* * * *

With regard to older patients, this claim which I am advancing (skilled examination of the larynx) has been insisted on by Sir Felix Semon, and he says that no person over forty should be allowed to remain hoarse for any length of time without a skilled examination, because he, like others of us, has been struck with the slow progress of epithelioma of the larynx in its early stages.

("Hoarseness," by StClair Thomson, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxx, p. 166.)

THE CLINICAL JOURNAL,

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ECTOPIA TESTIS.*

By W. McADAM ECCLES, M.S., F.R.C.S.

Gentlemen,—You are all familiar with imperfect descent of the testis—a by no means uncommon condition. Such imperfect descent is of two kinds: arrest and ectopia. Arrest of the testis is present when that organ is held up on its normal route on its way to the bottom of the scrotum. Ectopia testis is present when that organ has passed into an abnormal position, a position other than in the line of normal descent.

There are five such positions: perinæal, femoral superficial inguinal, cruro-scrotal, and pre-penial (Fig. 1).

There are only two causes of a transgression from the line of normal descent: the pull of lateral fibres of the gubernaculum attached to spots away from the normal, and the push of an advancing hernia. The gubernaculum testis certainly con-

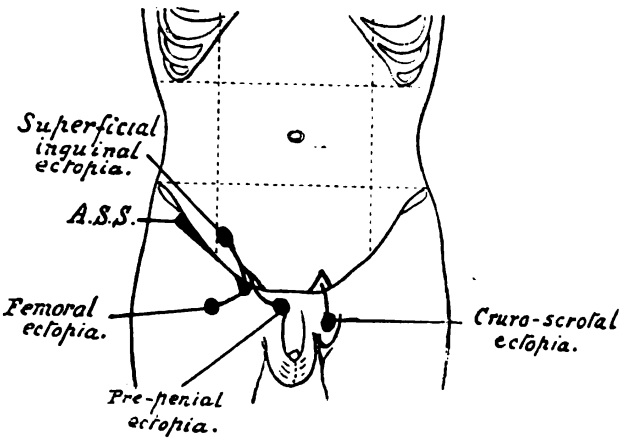

Fig. 1.—Diagram to show four of the common sites of an ectopic testis.

* A Clinical Lecture at St. Bartholomew's Hospital.

FIG. 2.—A left testis lying in the perinæum. Notice that the left half of the scrotum is developed.

FIG. 3.—Left testis in the perinæum. Notice the development of the left half of the scrotum.

tains unstriped muscular fibres, and sometimes fibres which show some striation. The lower attachments of the gubernaculum are chiefly into the tissue at the bottom of the scrotum, but fibres also pass into the perinæum to be attached to the inner side of the tuber ischii, others into the region of Scarpa's triangle, and others to the region of the root of the penis.

Supposing the pull of the perinæal fibres is strong, or the approach into the upper part of the scrotum narrow, it is easy to see why a testis passes along the route of greater pull or least resistance into the space between the anus and tuber ischii (Figs. 2, 3, and 4). This is the most common ectopic position, and is the normal position in the hog.

If it is the pull of the femoral fibres that is strong, and again the entrance into the scrotum difficult, the testis may be drawn over in Scarpa's triangle (Fig. 5). It is exceedingly doubtful whether the testis ever leaves the abdomen by the femoral ring, and enters the thigh by the crural canal. There are some cases reported of its having done so, but the evidence is not to my mind conclusive. I have in the course of operation

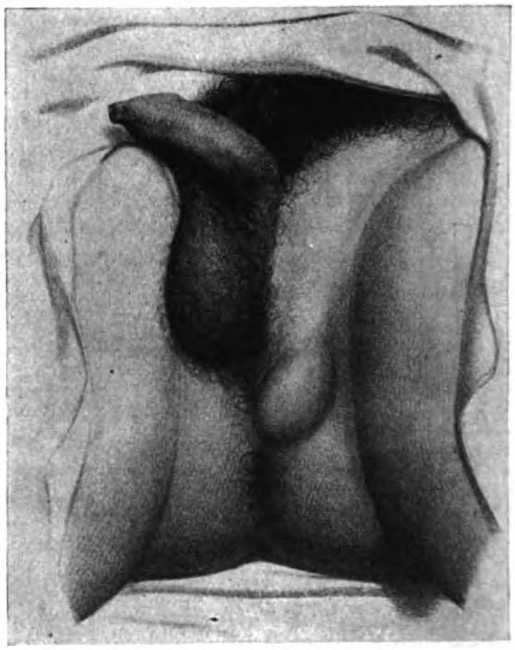

FIG. 4.—A left testis in the perinæum. Notice that the left half of the scrotum is represented as not developed.

FIG. 5.—A left testis in Scarpa's triangle.

for femoral testis exposed the cord on several occasions and found it emerging from the superficial (external) abdominal ring, but I have never seen it pass through the femoral ring.

Other attachments of the gubernaculum draw the testis to the root of the penis, and this position is the pre-penial, the normal position in the marsupials (Fig. 6).

If the peritoneal process which accompanies the testis into its abnormal position remains patent a congenital hernia may be present.

FIG. 6.—A right pre-penial testis. Notice that the right half of the scrotum is developed.

If a testis is found lying superficial to the aponeurosis of the external oblique muscle, it has reached this position by traversing the inguinal canal, by passing through the superficial abdominal ring, and then by being pushed by an accompanying and advancing hernia, the hernia for some reason being prevented from entering the scrotum.

In other cases the testis will be found in a pouch between the scrotum and the thigh, this pouch being covered by skin destitute of any dartos tissue (Figs. 7 and 8). This is the position of the testis known as the cruro-scrotal. A hernia by no means infrequently accompanies this ectopia.

The ectopic testis is not necessarily an imperfect organ. When in the perinæum it may be very fairly developed, and quite capable of producing spermatozoa. As a rule, however, in the other positions it is generally imperfectly developed as well as abnormally descended.

With regard to treatment, it is quite feasible and easy to transplant the perinæal testis into its own half of the scrotum; the testis has passed beyond its natural position, and its cord is therefore quite long enough to allow its being placed in the scrotum (Fig. 9). The femoral testis can also

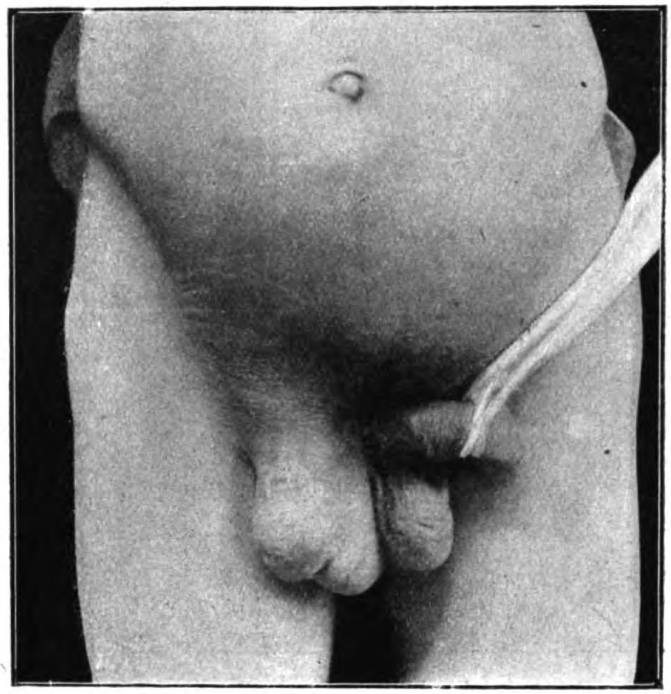

FIG. 7.—A right cruro-scrotal testis accompanied by hernia.

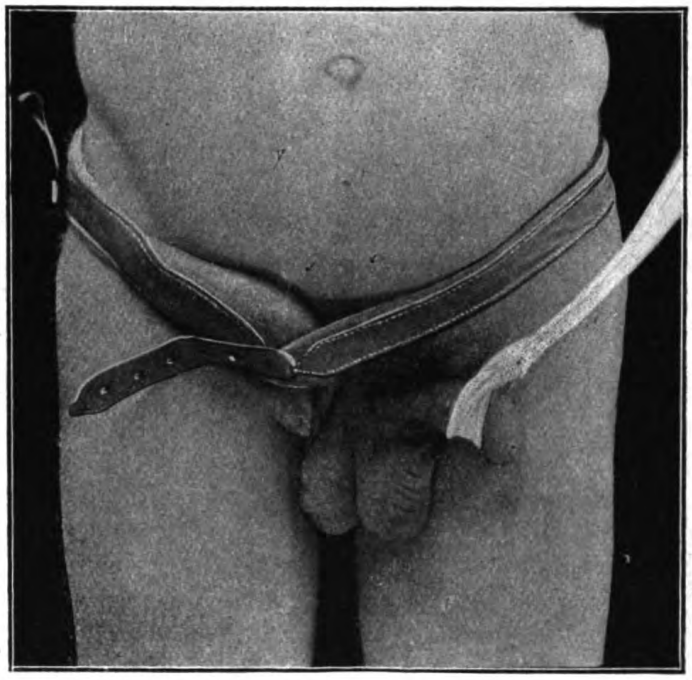

FIG. 8.—The same with the hernia retained by a rat-tailed truss. Notice the cruro-scrotal pouch distinct from the scrotum.

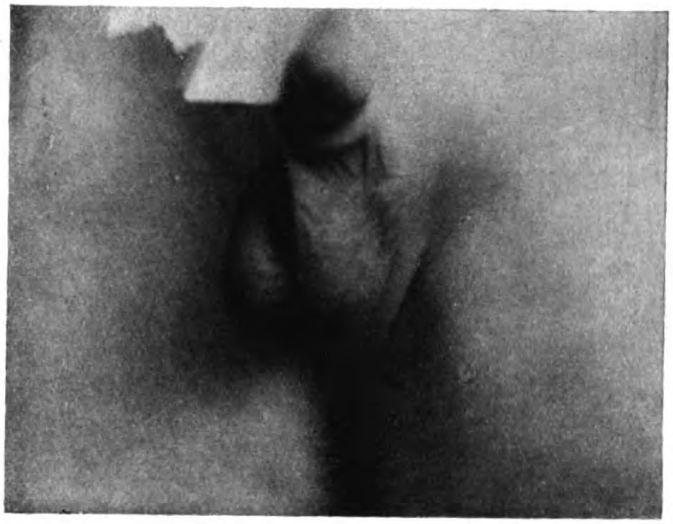

FIG. 9.—The left perinæal testis of boy, shown in Fig. 3, placed in scrotum by operation.

be transplanted fairly readily, and for the same reason.

The superficial inguinal, the cruro-scrotal and the pre-penial testis are usually poorly developed, and, especially when accompanied by a hernia, are best removed.

The ectopic testis is subject, like its normally placed fellow, to several diseases, particularly inflammation. The orchitis may be traumatic or infective. The abnormal position of the testis exposes it to injury. This is particularly the case with the perinæal testis. By no means infrequently the organ is subjected to pressure in sitting, especially on a bicycle saddle.

Inflammation of the perinæal testis simulates at times an ischio-rectal abscess, and peculiarly so when there is an infection by the gonococcus.

The mistake should not arise, for it can be avoided by seeing whether the corresponding half of the scrotum is distended by a testis or not.

An ectopic testis which is repeatedly the site of inflammation is best out of the way, the patient being rid of his trouble by the removal of the organ.

A hydrocele not uncommonly remains around the abnormally placed testis after an inflammatory attack. Treatment should be either the evacuation of the fluid, and the transplantation of the testis, or the excision of the testis with the tunica vaginalis containing the fluid.

A hernia does not accompany an ectopic testis so usually as it does an arrested testis. A hernia passing into the perinæal region along the same route as that followed by a perinæal testis may be called a superficial perinæal hernia, to distinguish it from the hernia which protrudes into the perinæal region through the pelvic floor.

If the sac contains intestine it presents as a resonant tumour lying between the anus and the tuber ischii, and can hardly be mistaken for any other tumour.

The superficial inguinal testis generally has a hernia associated with it—in fact, the hernia seems to force the testis onwards in front of it. It is because of this propulsion that the testis may come to lie actually external to the anterior superior spine of the ilium. When in such a position, or

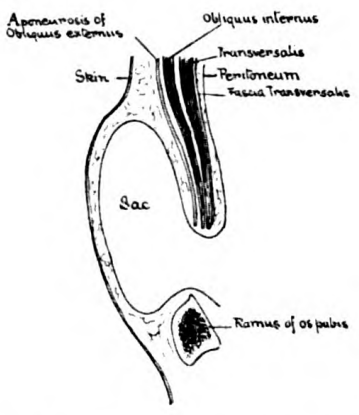

FIG. 10.—Diagram of a pre-aponeurotic hernia.

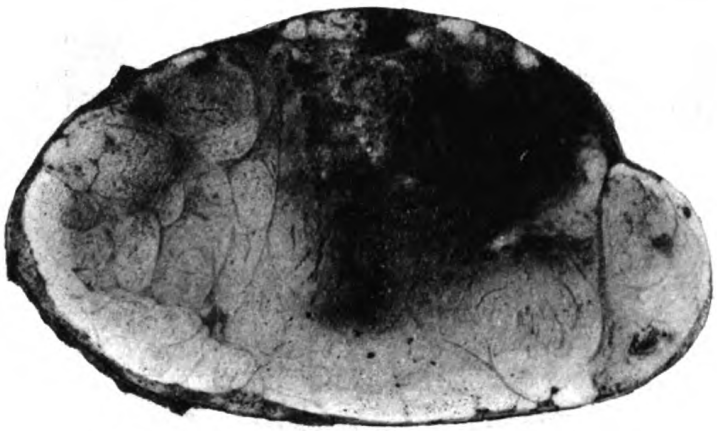

FIG. 11.—Macroscopic section of a sarcoma of a superficial inguinal testis.

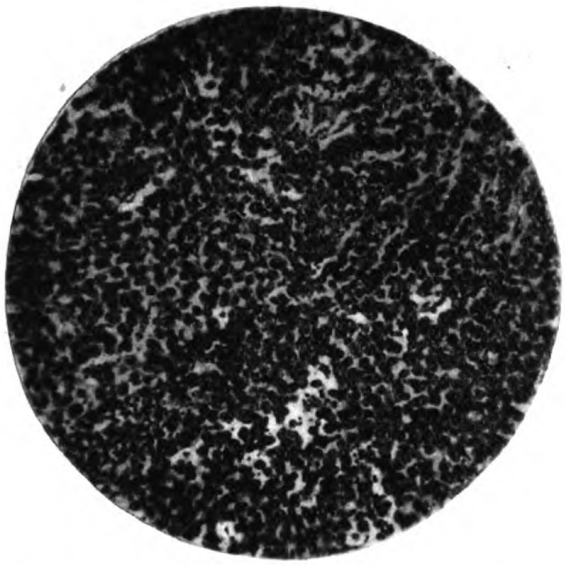

FIG. 12.—A microscopic section of the same sarcoma.

near it, the type of hernia is the pre-aponeurotic form of interstitial inguinal hernia (Fig. 10), in which the sac has passed down the inguinal canal, through the superficial abdominal ring, and then upwards and outwards in the plane of the abdominal wall between the skin and the aponeurosis of the external oblique muscle—in other words, in the subcutaneous tissues.

A cruro-scrotal hernia is one which lies between the scrotum and the thigh, and is covered with skin not containing dartos tissue. The corresponding half of the scrotum is developed, but not distended (Fig. 7).

When an ectopic testis is associated with a hernia an operation is always advisable, not on account of the testis, but for the cure of the hernia. As a rule the testis should be transplanted into its own side of the scrotum, if this is possible. Otherwise it is legitimate to remove it, a radical operation being performed upon the hernia at the same time.

Occasionally an ectopic testis becomes the site of a new growth, usually a carcinoma or a sarcoma (Figs. 11 and 12).

If this condition is even suspected, castration is urgently called for.

July 21st, 1913.

MINOR MALADIES AND THEIR TREATMENT. By LEONARD WILLIAMS, M.D., M.R.C.P. Third edition. Pp. 396. London: Baillière, Tindall & Cox. 5*s*. net.

The first edition of this book was published in 1906. The appearance of a third edition is the best recommendation of a work which has obviously become a favourite with the rising school of practitioners. The book might fitly be described as "points in practice," for it teems with suggestions in diagnosis and treatment, with salutary advice to avoid some not uncommon mistakes and pitfalls. A chapter on some skin rashes of common occurrence would be a welcome addition, as with that exception "the daily round and the common task" are completely covered.

A LECTURE

ON

ENLARGEMENT OF THE SPLEEN.*

By FREDERICK TAYLOR, M.D., F.R.C.P.,
Physician to the Hospital, and Consulting Physician, Guy's Hospital.

GENTLEMEN,—I have chosen for the lecture to-day a subject which should be familiar to you, but on which there are still some points which possess some interest. The first question to be raised in connection with enlargement of the spleen is as to the method of measuring or estimating the degree of enlargement, or for concluding whether it is enlarged or not. As you know, the spleen is situate in the left hypochondrium, at the outer side of the fundus of the stomach, and in health it is entirely covered by the ribs. It is a thoracic organ in that it lies within the bony thorax, but it is an abdominal organ in that it is below the diaphragm. Enlargement of the spleen, with rare exceptions, takes place in a downward direction. Normally it lies underneath and against the ninth, tenth and eleventh ribs. The tip of the eleventh rib, you will remember, reaches to the mid-axillary line. As a rule the spleen does not reach down to the costal margin; in other words, it cannot be felt in the ordinary way at the costal margin unless the patient take a deep inspiration, and so pushes it down to that line. The area over which you can elicit dulness will vary with the degree of distension of the stomach, and an emphysematous lung can overlap the upper and posterior part of the spleen. The limiting line for the anterior and lower border of the spleen has been given differently by various authors. A line, which many writers have given, is the mid-axillary line or lateral line of the body. The line I have always been in the habit of adopting, and which I believe is the best, is a straight line drawn from the left nipple to the tip of the eleventh rib. It has this advantage over the lateral line, that it is not a vertical line, but one which runs downwards and backwards, and is, as nearly as possible, perpendicular to the longitudinal axis of the spleen, and to the direction in which the spleen enlarges. If you percuss accurately over a well-marked spleen, and attempt to take the lateral line as its anterior limit, you will find that the splenic dulness reaches in front of it, whereas if you take this other line, from the nipple to eleventh rib, you find the anterior and lower border of the spleen is above and behind it. Some teachers have adopted a line drawn from the mid-clavicular point down to the tip of the eleventh rib. The objection I have to that is that it requires an effort to get at the mid-clavicular point, because the clavicle is continuous with the acromion process. You must first find the acromio-clavicular joint before you can measure the clavicle. And, moreover, the line which you draw is not sufficiently oblique to give the advantages presented by the line from the nipple to the eleventh rib line.

In different people the spleen is not absolutely of the same size, but I think it may be presumed, in any case in which you have reason to think it likely that the spleen will be enlarged, that if you find an area of dulness in front of that line, that is, over the space about 1½ inches broad between that line and the costal margin formed by the ninth and tenth ribs, there is some enlargement of spleen. If the enlargement is greater, the organ comes down below the costal margin. Your observation will always be assisted by making the patient take a deep inspiration, if he is in a position to do so, because the spleen is a movable organ, and it is moved by the contraction and descent of the diaphragm. In any case, unless the spleen is adherent, you get the spleen moving down one inch below its upper position near the costal margin, and when it is enlarged it almost necessarily comes below the costal margin. The enlargement takes place in a downward and forward direction, and in different cases of enlargement of the spleen it may reach far down into the abdomen, extending forwards also towards the middle line, and reaching down to Poupart's ligament, or the crest of the ilium, or the anterior-superior spinous process. When enlargement proceeds to an extreme degree, as it does in some diseases, which I shall mention presently, the spleen takes on a curved, or almost semicircular form, bulging the left flank above, working round above the pelvis in close contact with the pubes, and reaching the middle line, or crossing two or three inches to the right of the middle line, below the umbilicus. That is characteristic of extreme splenic enlargement. In these

* Delivered at the Seamen's Hospital, Greenwich.

circumstances the upper part of the spleen rarely reaches to the middle line above the umbilicus though it crosses it below. And, as was long ago pointed out, there is very little doubt that this is due to the arrangement of the splenic vessels. The splenic artery arises from the cœliac axis, and the arteries are links or bands which retain the spleen. And as the organ elongates downwards it follows the circumference of a circle of which the cœliac axis is the centre, and the branches of the splenic artery are radii.

The spleen does not, as a rule, enlarge in an upward direction, simply because in the greater number of cases of splenic enlargement the enlargement is uniform, and it finds less resistance in a downward direction, and is, moreover, in that direction assisted by gravity. In the case of the liver, also, the greater number of enlargements are in a downward direction. If there is an enlargement of the liver upwards into the chest, it is due either to localised cancer in the upper part, or to an abscess, or to a hydatid cyst. In the same way we may take it that the spleen enlarges, and as cancer and hydatid of the spleen are much less common than the same diseases in the liver, it is not surprising that upward enlargements of the spleen are comparatively rare.

We may now consider what are the other characteristics of the spleen as it is in its normal position. Let us take one of moderate size. There is a mass felt immediately under the parietes; it is dull, never resonant; the mass is in close contact with the abdominal parietes, and all the hollow viscera, like the stomach, colon and intestines which are near it, are always on the right side, or the deeper side of the organ. The mass which you can feel under your hand can be traced upwards as far as the costal margin, and appears to pass under the costal margin. The dulness over it is continuous with the upper area of dulness to which I have referred. Very generally the organ is smooth. If there are masses of cancer, or of lymphomatous growth, or cysts within it, the surface may be rough, but these are comparatively rare. The organ, as a rule, unless it has reached an enormous size, moves on inspiration, for it is in contact with the diaphragm above. It may, if it is very large, come into contact by its lower limits with the upper borders of the pelvis, so that it cannot easily move downwards. But when it is less large, and the lower end is free in the abdomen, a deep inspiration will drive it down from 1½ to 2 inches.

We may consider for a moment the diagnostic importance of the movement of the different organs in the abdomen during the action of the diaphragm. The organs which are in contact with the diaphragm are necessarily driven down by the diaphragm when that muscle contracts. The liver is driven down by inspiration an inch or more. The spleen is driven down, whether it is enlarged or healthy. In the diagnosis of malignant growth of the pylorus we know that unless the growth has formed considerable adhesions to surrounding parts, the palpable mass is driven down by inspiration, since the stomach is in contact with the diaphragm. And the kidneys, which lie in the upper and posterior part of the abdomen in contact with the diaphragm, also descend with deep inspiration. Do other organs in the abdomen behave differently on inspiration? The transverse colon lies immediately under the lower border of the stomach. It is clear that if the stomach is driven down the transverse colon must be moved to a certain extent, or the contents, gaseous or liquid, of either or both must be compressed. If there is a solid mass or growth in the transverse colon, the compressibility of the contents of the stomach may prevent much motion being communicated to it; and in any case this motion might be checked by the mesocolon attaching it to the back of the abdomen, so that the movement would not be so extensive in the case of a mass in the colon as it would in connection with a mass in the pylorus. An aneurysm of the aorta would not be moved much by the descent of the diaphragm. On the one hand the hollow organs would be compressible between the diaphragm and the tense aneurysm; on the other, the aneurysm fixed by its base to the aorta could only yield a little at its anterior portion to a force coming from above. A growth in the ascending or descending colon is probably fixed by various adhesions, but if not, may receive some downward impulse from contact with liver or spleen. So, in the main, the organs which are in contact with the diaphragm directly are moved to a certain extent by the diaphragm. The other parts, which are not in contact with the diaphragm, and especially those which have connections which fix them to the posterior wall, are moved very little.

There is another feature of the spleen to which one must refer, and that is the presence of a notch in its anterior border. This cannot always be observed, and it never is until the spleen has descended well into the abdomen. Even then you must be prepared to meet with cases in which you have to recognise the spleen, and yet in which you do not feel a notch. A notch (or notches, for there may be two) can best be observed when the organ is quite large, and where it is curved round in the way I have described. After what I have been saying, one might think that there could not be any difficulty in discovering that a spleen was enlarged, or that any mistake could be made as to its identity, where the mass was of considerable size.

Still, difficulties do arise. I remember a mistake which was made, not, it is true, by a skilled physician, but at an examination of candidates for diploma, in the case of a man who was very muscular, on the surface of whose body one could see an outline which was, roughly, spleen-shaped. It was really an unusually developed external oblique muscle, but this candidate regarded it as a spleen. It was part of the abdominal parietes, and had a thick round edge. What should have been remembered was that you do not generally see the outline of the spleen through the parietes, though it can be felt there.

The kidney is just behind the spleen, and if the kidney enlarges very much it forms a mass in the left flank, and you must think whether you could mistake it for a spleen. Could you mistake a hydronephrosis for a spleen? I think possibly you could. The points which should guide you are these: I have told you the spleen is always close to the parietes, and the edge can generally be distinctly felt. No part of the bowel, small or large, ever comes in front of it. The colon is in front of the kidney normally, and with a moderate enlargement of the kidney, from whatever cause, the resonance of the colon may still be recognised in front of the mass, at least at its inner border. Only with increasing size of a renal tumour or hydronephrosis the colon may be forced inwards; but the renal tumour will never present the edge and notch and concave outline which the largest spleens so commonly show.

When I was speaking of the organs which might be driven downwards by the diaphragm, I left out of consideration the pancreas. The pancreas runs across the aorta, and the spine behind; and if it is in contact with the crura, it cannot be considered to be in close contact with the fibres of the diaphragm which contract on the abdomen. But there may be a pancreatic cyst, and that cyst may present itself in the epigastric region; and if it extends to the left there may be considerable difficulty in being sure from what organ the tumour concerned arises. I have known the difficulty arise once or twice. A rounded tumour in the epigastrium extending to the left costal margin and nearly down to the umbilicus certainly descended on inspiration. Because of its extensive encroachment over the middle line, I felt sure it was not a spleen. But it turned out to be a pancreatic cyst, a possibility which we did not consider very fully, and which, I think, then I should have rejected, because of its very free movement in a downward direction. A few years back we had almost the same condition of affairs in a patient in this hospital under one of my colleagues. I was going over it in the same way, and practically the same position of affairs arose. There was a globular mass, and one could not feel fluctuation distinctly. The mass moved downward on inspiration to a distance of about 1½ in., and much difficulty was felt as to its accurate diagnosis. Remembering my former experience, I admitted the possibility of its being a pancreatic cyst. Eventually it was operated upon, and proved to be so. So remember that, even though the pancreas itself, lying back and not in contact with the arching fibres of the diaphragm, may be little affected by inspiration if the patient has a pancreatic cyst sufficiently large, and extending forwards into the abdomen, it may be moved by the diaphragm. Thus this property of descent on inspiration does not belong exclusively to the five organs I have named.

A malignant mass in connection with the colon may be confounded with the spleen. It is not likely to be moved down by a deep breath in the same way that the spleen is, nor is it likely to be perfectly smooth on the surface; nor to be in close contact with the anterior parietes, though it may be on the posterior.

Then there is the possibility of the mistake, which no doubt is rare, but which has been made

more than once. I refer to mistaking a spleen for an ovarian tumour.

You may say it is difficult to see how this could happen; but I think it is explained somewhat in this way. The disease leucocythæmia is sometimes so insidious that the spleen can reach an enormous size before the patient consults a medical man. There is then found a unilateral mass, which extends from the pelvis to the costal margin. The mass is wrongly regarded as arising from the pelvis instead of the thorax, and the important fact that an ovarian tumour of such a size would have already taken a central or bilateral position is overlooked. But it might almost be said that now such a mistake is impossible, and that the above remarks have a historical rather than a practical interest.

I now come to the question of causes of enlargement of the spleen, and they are numerous and interesting. And yet they can, for the most part, be grouped very definitely. And the first thing, which we are all familiar with, is that one of the chief causes of enlargement of the spleen, though generally in rather moderate degree, is some form of infection. We might almost say that enlargement of the spleen may occur in all infections. In typhoid fever it is well known that the spleen enlarges to a moderate degree, and it is a diagnostic feature. But if other infections are accompanied with enlargement of the organ, it cannot be pathognomonic of typhoid, and it is not. But it is useful if you have to diagnose typhoid fever from tubercular meningitis, because the latter, although also an infection, does not lead, as a rule, to such an enlargement of the spleen as does typhoid. In relapsing fever, again, the spleen is constantly enlarged, and it is frequently so, too, in malaria, and in chronic malarial infection there is a mass of considerable size. We may associate with these the great enlargements of the spleen in some tropical infectious diseases, such as kala azar, and an allied, but apparently not identical disease, occurring in Egypt. Another striking instance is the infection of malignant endocarditis, in which, although infarcts usually occur in the spleen, which may be thought to contribute to the enlargement, the chief cause of enlargement of the spleen is the fact that there is a general infection.

That is one group of causes. At the same time we must recognise that there are many infectious diseases in which one cannot recognise the enlarged spleen clinically, and one, viz. yellow fever, in which it has been said to be never enlarged.

Allied to the infectious group is the lardaceous spleen, in which the enlargement is due to a deposit of unorganised material, the result of chronic infection, in most cases by pyogenic organisms, or by the spirochæta of syphilis.

A second group is the obscure group of diseases of the blood, in which we have some of the most extensive enlargements of the spleen. I have already figured the spleen which is characteristic of an advanced case of leucocythæmia. There are two forms of leukæmia, the medullary or myelocythæmic, sometimes called myelæmic, and the lymphatic or lymphæmic. In the one case there are a large number of myelocytes in the blood, and in the other case, the lymphocytes, large or small, increase enormously and form from 90 to 95 per cent. of the white corpuscles. In both those conditions there may be an enlarged spleen; it is not, however, so frequently of great size in the lymphæmic cases as in the other form, but it may reach six or eight inches below the costal margin even in the former. In Hodgkin's disease the spleen is enlarged, sometimes considerably, in association with enlarged lymph-glands, but without any material alterations in the blood. Then we have the condition known as splenic anæmia, in which there is often great enlargement of the organ. Then there is pernicious anæmia, whether it forms a single disease or a group with many varieties which is accompanied with moderate splenic enlargement. Another disease, of which we had a very good instance at this hospital not long ago, is somewhat rare—acholuric jaundice, a jaundice without bile in the urine, and with a condition of blood nearly characteristic of pernicious anæmia. When I first saw the case I spoke of it as one of pernicious anæmia, with jaundice, as there was a yellow tint of the conjunctiva. But it was not the lemon-yellow colour which is so characteristic of pernicious anæmia, and we grouped it finally into the class of acholuric jaundice, which occurs sometimes as a congenital family disease and sometimes as an isolated acquired adult disease. This case was one of the latter class.

Another condition in which you find the spleen enlarged is infantile pseudo-leukæmic anæmia.

The patients are infants, or small children, who have anæmia and enlarged spleens, and a condition of blood in which the myelocytes are present to a slight extent and there is a moderately free leucocytosis.

In children there are many conditions besides this in which the spleen is enlarged, either diseases of the blood or infective conditions, as, for instance, infantile syphilis and rickets.

Those are some of the conditions in which you find enlargements of the spleen. The diagnosis is arrived at by a careful consideration of the state of the blood. The leukæmias are distinguished by the excess of leucocytes and the differential count; the great number of myelocytes in the one form, and the extraordinary predominance of lymphocytes in the other. Splenic anæmia is characterised not only by the small number of red corpuscles, but especially by the small number of white corpuscles (so-called *leucopenia*), which are reduced from the normal 7000 or 8000 to 3000 or 4000. These cases, like other cases of blood disease, are frequently accompanied by severe hæmorrhages. In the more familiar form of pernicious anæmia the excess of hæmoglobin in relation to the number of the red corpuscles is regarded as a distinctive feature, and the leucocytes vary little from the normal. In acholuric jaundice the colour-index may also be high.

Another disease whose pathological relations are very uncertain is splenomegalic cirrhosis. In these cases, cirrhosis of the liver has occurred in association with an unusually enlarged spleen, which seems too large to be the result of portal congestion, but is more probably due to infective processes in connection with the cirrhosis.

We have splenomegaly or enlargement of the spleen in yet another association, and that is with polycythæmia. In these cases the red corpuscles are enormously increased up to eight, nine, or even ten millions, instead of the normal five millions, a condition which is the very opposite of that which was in so many other forms of splenic enlargement; and I think it must be admitted we are still very far from a thorough comprehension of the way in which the enlargement of the spleen comes about in the different cases.

I have still to remind you that there are adventitious causes of enlargement of the spleen, such as cancer and cysts. But they are quite infrequent, and they would be almost at once recognised by the outline of the organ if this were sufficiently enlarged to come fully under examination. With the exception of these last cases the organ always retains its normal outline: it is a big spleen, with a comparatively smooth rounded surface, with an anterior or internal edge; and the larger it gets the more likely is it to present one or more notches to the examining hand.

It is not my intention—and indeed there would not be time—to go into the histological conditions and changes which take place in the spleen in these blood diseases; there is still much to be learned about them. But in nearly every case in which there is considerable enlargement there are infarcts in the spleen. These necrotic patches are characteristic of malignant endocarditis, because from the fungating or ulcerating surface particles may be dislodged, and may be carried into the spleen, and cause the local anæmia and necrosis which constitute an infarct. But it is equally true that in many of these diseases of the blood infarcts occur, though there is no endocarditis and no evidence of particles being conveyed into the arteries. They occur in splenic anæmia, in leukæmia, in acholuric jaundice, and in splenomegalic jaundice. And in the enlarged spleen which we took from our patient with acholuric jaundice a few weeks ago, such infarcts were present.

The treatment depends on the nature of the lesion. In some of these diseases the splenic enlargement appears to result from the general condition of the blood and bone-marrow, and we know that in leukæmia the removal of the spleen has been useless.

But in some cases splenectomy has been successful, and followed by restoration to health, for instance in splenic anæmia and in some cases of acholuric jaundice. But the pathology of these cases is still too obscure to allow us to say with confidence in what way and to what extent the spleen can be regarded as the *fons et origo mali*.

July 21st, 1913.

SOME REMARKS UPON THE DIAGNOSIS AND TREATMENT OF LACRIMAL AFFECTIONS.*

By SYDNEY STEPHENSON, D.O.(Oxon.),
Ophthalmic Surgeon to the Queen's Hospital for Children, London, and to King Edward Memorial Hospital, Ealing, W.

THE last few years have witnessed a great change in the methods of treating affections of the lacrimal passages. When I was a clinical assistant, every day part of our duty was to pass lacrimal probes upon patients who had suffered from epiphora, often for years together. The operation was unpleasant for all concerned. Local anæsthesia was not so well understood as it is nowadays, and doubtless the technical skill of some of us left a good deal to be desired. The disheartening part of the whole business was that so few of the patients were cured.

Mais nous avons changé tout cela. Now, when a patient consults us on account of a watery eye, we do not at once assume, as we formerly did, that the underlying cause must be an organic stricture of the nasal duct. A surgeon first eliminates such obvious causes of epiphora as a displaced or occluded punctum lacrimale or a streptothrix obstruction in the canaliculus, and he then examines the nose and remedies any obvious defect, such as chronic nasal catarrh, due to this or that cause. Many cases of epiphora are associated with hypertrophic rhinitis, and can be cured only by attention to that condition.

No obvious cause having been found to account for the symptom, the surgeon would drop a little fluorescein into the conjunctival sac, and, after waiting for a few minutes, would see whether any of the dye had passed into the nose. For this purpose he would inspect the nasal chamber, by the aid of a speculum, or, alternatively, would get the patient to blow the nose, in order to ascertain whether traces of the stain could be found on the handkerchief. If the lacrimal passages are permeable, that will almost certainly be the case. This simple test, in my opinion, is preferable to diagnosis by syringing the passages, inasmuch as the latter, when it succeeds, merely proves that fluid under pressure can pass, which is not quite the same thing as fluid passing under normal conditions.

In cases where there is no definite evidence to be obtained of organic obstruction in the nasal duct, the treatment of the patient is commenced with antiseptic collyria, such as argyrol, 10 per cent., or protargol, 5 per cent. Argyrol penetrates with singular facility into the lacrimal sac and inferior meatus of the nose. Inspection of the back of the throat in those who are using argyrol to the eyes often shows traces of the product on the mucous membrane of the parts. If the mucous membrane of the nose appears to be unhealthy, some simple lotion, such as Dobell's, should be prescribed, and directions should be given that a small quantity is to be sniffed up by the patient two or three times a day. This can do nothing but good. Another plan which I have known succeed is to paint the affected mucous membrane daily, or at less frequent intervals, with a 2 per cent. solution of silver nitrate.

If, after several weeks' employment, these several measures fail to relieve the epiphora, the surgeon resorts to the lacrimal syringe. After dilating the lower punctum (if it be too small to admit the nozzle of the syringe otherwise), he passes the point of the instrument along the lower canaliculus into the lacrimal one, and then endeavours slowly to wash out the sac and duct with tepid saline (1·4 per cent.), or boric lotion (10 grs. to the ounce). This may not be possible at the first few trials, the injected fluid regurgitating into the conjunctival sac through the upper canaliculus and punctum. But, *provided the condition has not been treated by probing*, the plan is likely to succeed sooner or later, and eventual cure is then not far away.

It would seem a practical method of dealing with dacryocystitis to syringe the lacrimal passages with argyrol or protargol; and so, in point of fact, it is, provided that certain precautions be taken. The little operation should be undertaken with circumspection. No force should ever be used, and fluid should not be injected when there exists any reason for suspecting a recent abrasion of the mucous membrane, such as might easily follow slitting or dilating the canaliculus. Under these circumstances very serious accidents have hap-

* Part of a Clinical Lecture delivered to the candidates for the Diploma in Ophthalmology in the University of Oxford, June, 1913.

pened. Thus, in a case mentioned by Elmore Brewerton ('The Ophthalmoscope,' April, 1906, p. 206), orbital cellulitis, with total loss of sight in the affected eye, resulted from injection into the lacrimal sac of a 33 per cent. solution of protargol, compounded one hour previously.

A more trivial incident, but all the same one to be carefully avoided, is staining of the tissues of the eyelid and orbit. For example, Post reported a case where, after the injection of a 25 per cent. solution of argyrol into the cavity left by the removal of a chalazion, the entire lid became black and shining, like "a highly polished stove-lid," to quote his graphic simile '(Ophthalmic Record,' July, 1905). Lastly, the reader need scarcely be reminded that prolonged treatment with any of the preparations of silver, organic or otherwise, may lead to permanent staining of the conjunctiva (*argyrosis*).

In view of the success of the various steps described above, we are bound to assume that organic obstruction of the naso-lacrimal duct is not so common as we once believed. The truth of the matter seems to be that a chronic dacryocystitis does not necessarily imply a strictured naso-lacrimal passage. It now appears likely that a stricture may be secondary to a primary inflammation of the lacrimal sac; and we know positively that in many cases it has been manufactured by the injudicious passage of the lacrimal probe—in other words, the obstruction may be the result and not the cause of the dacryocystitis. The common condition—that relieved by the use of collyria or the syringe—is essentially a catarrh dependent upon a similar condition of the mucous membrane of the nose. Relatively slight catarrhal changes may occlude the lumen of the nasal duct, more especially as a venous plexus, liable to periodical engorgement, lies immediately beneath the mucous membrane. In this way alone can we account for a statement not seldom volunteered by patients, namely, that the watering of the eye is sometimes absent for several days together. In a number of cases dacryocystitis is associated with, and caused by, changes in the posterior ethmoidal cells or antrum.

Yet there remain a few patients, more especially in the hospital class, whose condition does not materially improve under the measures described. How are they to be treated? If there be any obvious disease of the nasal passages beyond the help of simple remedies, a wise eye surgeon will transfer the cases to his rhinological colleague. The removal of an hypertrophied turbinate bone or of a polypus, or the straightening of a deflected septum, may cure a watering eye.

As to patients who appear to suffer from an organic stricture of the nasal duct, as shown by the fluorescein test, and the failure of ordinary treatment, there remain three methods of dealing with the case: (1) probes, (2) extirpation of the lacrimal sac, and (3) Toti's operation.

(1) The first method is still adopted by the more conservative ophthalmic surgeon, despite its very obvious disadvantages, and the fact that 10 per cent. of the cases at the outside are permanently relieved.

If we decide to treat the condition by the probe, a first indication is to secure adequate local anæsthesia. This should consist in no mere perfunctory dropping of a little cocaine into the conjunctival sac. On the contrary, careful steps should be taken to render the passage of the probe as little obnoxious to the patient as possible. To begin with, the inferior meatus of the nostril corresponding to the affected eye should be plugged with a morsel of absorbent cotton moistened with cocaine (4 per cent.). This is best introduced, under the guidance of a mirror, by means of nasal forceps. In the next place, we drop a 2 per cent. solution of cocaine over the inferior punctum and conjunctiva, and by gentle massage endeavour to aid its absorption. Lastly, by means of a syringe, we fill the lacrimal sac and the naso-lacrimal duct, as far as may be, with sterile oil containing cocaine (2 per cent.), and adrenalin (0·1 per cent.). If these steps be taken, the operation may be performed practically without pain, with the further advantages that the mucous membrane will be contracted by the adrenalin, and the passage of the probe facilitated by the oil that has been injected into the drainage apparatus. Bowman's probes, of gradually increasing calibre, may be passed at intervals, until those of medium size may be readily introduced. The treatment is then discontinued for a time; but, in my experience, there is apt to be recurrence sooner or later, when probing may be undertaken again, or the case treated by the measures to be next described.

(2) The modern tendency in such cases is to remove the lacrimal sac, and the results of that operation, as a rule, leave little to be desired. Contrary to expectation, the eye waters little after successful extirpation of the sac. This is explained by assuming that the secretion of tears is increased by the reflex stimulation of a diseased lacrimal sac, and that when the last-named is removed, the secretion is no longer excessive. A tendency to epiphora after operation is easily remedied by removing the accessory lacrimal gland, and some surgeons, especially in Germany, even make a point of taking away that structure at the same time as they remove the lacrimal sac. The operation is quite simple, and usually succeeds in curing any epiphora that may be present.

The great value of extirpation of the lacrimal sac, as it seems to me, is when dealing with organic obstruction in the working classes, who are particularly subject to the malady. They can seldom afford the time necessary for treatment by probes, and, often enough, of persevering with that uncomfortable and inefficient method of treatment for any prolonged period. Furthermore, prompt removal of the diseased sac, as insisted upon by Axenfeld, minimises the danger, otherwise run by this class of patient, of developing that destructive disease, ulcus serpens of the cornea (*hypopyon-keratitis*), liable, in presence of a suppurating sac, to follow any injury to the cornea, however trivial.

Another absolute indication for removal of the sac is to be found in tuberculous disease of the sac—a condition that, in my personal experience, is not altogether uncommon in children. In that disease, the passage of probes should be utterly condemned, since they are likely to do harm and not good.

(3) Toti's dacryocysto-rhinostomia is a modern substitute for extirpation of the lacrimal sac. It aims at securing a wide communication between the cavity of the lacrimal sac, on the one side, and the nose, on the other. By resection of the nasal wall of the sac, it converts the sac into a part of the nasal cavity. In order to secure free drainage it is usual to resect the middle turbinated body, and this is commonly done before the Toti operation. The drawbacks to the operation are that the window made in the mucous membrane may close, thereby nullifying the effects of the intervention, and, further, that epiphora may not yield to the operation, although no discharge can be obtained by pressure upon the parts. But, according to Dr. Josef Meller, of Vienna, complete restoration of normal lacrimal drainage follows in about one-half of the cases treated by Toti's operation ('Ophthalmic Surgery,' second edition, 1912, p. 35).

So far as I know, no cases have yet been published by English ophthalmic surgeons, although Mr. Charles Higgens demonstrated Toti's operation at the Oxford Ophthalmological Congress two years ago.

July 21st, 1913.

Migration of a Fractured Rib.—Molco describes the case of a stout woman who was knocked down by a carriage which passed over the right side of the body. When admitted to hospital on the following day she complained of pain in the abdomen, the anterior wall of which showed extensive ecchymoses. There was no peritonitis or evidence of visceral injury, and the thorax and its contents appeared normal. On careful palpation of the abdomen some days later a foreign body was felt close to the right anterior superior spine, and half an inch above the crest of the ilium. This was a hard rectangular object 3 or 4 cm. in length situated in the subcutaneous fat, its long axis being in the direction of a line joining the pubis and the right lower ribs. It could be palpated and moved about without causing pain. On cutting down on this body it was found to consist of a fragment of rib 3 cm. long and was easily removed. A more careful examination of the chest showed that the right twelfth rib was shorter than the left, and appeared more freely movable. The mechanism by which this unusual displacement was produced is obscure, but the writer suggests that the fracture was produced by the first impact of the wheel of the carriage, and the fragment lying with its long axis in the line taken by the wheel in passing over the body of the patient, the portion of bone was driven before it through the subcutaneous fat on the abdominal wall to the place where it was found.—*La Presse Medicale*, p. 420.

SOME REMARKS

ON

GOUT, RHEUMATISM, AND RHEUMATOID ARTHRITIS.

By T. M. ALLISON, M.D.,

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In medicine the personal equation is a factor which we cannot estimate. Of two people with phthisis or influenza, the personal factor determines the fate of the sufferer in one case, whilst the severity of the attack perhaps determines it in the other, and these two people may apparently be of equal size, weight and strength, and the severity of the disease may in both cases appear to be equal. The chief certainty of medicine is its uncertainty, and therefore it is too often speculative rather than positive. But when one can be positive it is a great delight, and I wish to be so in this paper.

Gout.

In gout there is a distinct facies, a chalky-white complexion, which in a person of forty is unmistakable. Gout only occurs in beer- and stout-drinkers, *i. e.* when of alcoholic causation. Beer sets up fibrosis of the kidney, and renders it incapable of excreting uric acid, and gout is the result. Whiskey attacks the liver, beer and stout the kidneys. It is the constant moderate or immoderate drinkers who suffer, as for example the washerwoman who takes one to two bottles of beer daily, for twenty to thirty years. The people who get the most good out of alcohol are the teetotallers and the intermittent drinkers. The regular drinkers of small or large amounts of alcohol suffer more physically than the weekly drunkards. Small doses, especially of beer, fall on the kidneys, large doses, especially of whiskey, on the liver. The constitution should not be under the *constant* influence of alcohol, however small the daily amount.

The clinical features of gout are, therefore, usually associated with a history of beer-drinking or of lead intake over a period of time. If a patient comes to you with a history of joint pains, a look at the chalky white complexion and the presence of tophi in the rims of the ears faithfully foreshadow the diagnosis. If the condition commenced in the big toe of either foot the diagnosis is sure. If the feet or hands are moderately affected, the irregularity and hard nodular outline of the uric acid deposits in the joints strike one in comparison with the spindle-shaped, solidly œdematous feeling of the joints in rheumatoid arthritis. Both diseases attack the smaller joints—rheumatism the larger ones. Rheumatoid arthritis also is fonder—much fonder—of the wrist than gout. I do not remember to have seen a gouty wrist. The best treatment of gout is citrate of lithia in 10-gr. doses thrice daily.

Rheumatism.

True rheumatism only occurs in mouth-breathers. There are two kinds of mouth-breathers: (1) the diurnal and nocturnal, and (2) the nocturnal only. Many people who can breathe comfortably through the nose during the day open and breathe through the mouth at night. It would therefore be clearer to say that true rheumatism only occurs in people who snore. It is a microbic disease entering through the tonsils, rheumatic pains being caused by toxin, and acute rheumatism (rheumatic fever) by germ and toxin combined. The pains, made worse by changes in the weather and by the cold air of the bedroom, are accounted for by the rheumatic sufferers being mouth-breathers. The warmth and rest of bed should ease rheumatic pains; they are worse because the cold air of the bedroom affects the fauces on coming from a warm fireside, and with consequent absorption of rheumatic toxin the pains increase. The facies of the rheumatic is characteristic. It is the face of ruddy health. This may be modified, but is not overcome, by the pallor of mitral mischief. The open mouth takes in a larger amount of oxygen, but permits invasion of the tonsils at the same time. Rheumatism occurs in young, healthy-looking subjects with a breach in their first line of defence. Children who are attacked are those affected by enlarged tonsils and adenoids, and rheumatism is hereditary chiefly because mouth-breathing is hereditary.

The best treatment of rheumatic pains is a gargle of—Acid carbolic liquidum, 3iv; Aq. chloro-

formi ad., ℥viij. Ft. gargle and tooth-wash—℥j in a cup of water. Used frequently.

This will often of itself abort an attack of sciatica when salicylates internally fail. And in acute rheumatism this gargle with Amm. bromide, gr. viij; Sod. salicyl., gr. xx.; Spt. æth. nit., ♏ x; Liq. ammon. citratis, ℥j; Aq. menth. pip. ad, ℥ss. ℥ss every two or three hours in water, with a preliminary purge, is almost specific. The salicylic treatment should be pushed, and the patient warned of the tinnitus aurium which will be produced. Then, and not till then, should the frequency of the doses be diminished.

The hyperpyrexia of acute rheumatism is due to salicylic acid poisoning when the kidneys fail to excrete properly. The urine of all cases of rheumatism should be examined for albumen, and the amount of urine excreted daily should be carefully watched. The constipating effect of the salicylates should also be counteracted by purgatives. The rest of the clinical picture of rheumatism, its painful attacks on the larger joints, the relaxing of its hold on one joint to seize another, the sour-smelling sweat, and the crippling of the cardiac valves and muscle are given in all text-books of medicines. But one point needs emphasis: if you cure mouth-breathing you prevent rheumatism, and prevent what is much worse—valvular disease of the heart. There will be much less heart mischief in future owing to the work of our throat specialists amongst children.

Rheumatoid Arthritis.

Rheumatoid arthritis has, like gout and rheumatism, a distinctive facies. It produces a pale and nervous face—quite distinct from the chalky-white complexion of gout or the ruddy hue of rheumatism. Its pathology is unknown. It may be due to germ or toxin, though I have tried joint after joint in vain for an organism. Some cases may be due to intestinal absorption, as a half-drachm dose of sulphate of magnesia in the morning has cured one of the only two cases I have seen benefited by medicine. The spindle-shaped, boggy joints are very characteristic, whilst the wasting of the muscles in the neighbourhood of the joints—especially of the wrist—the glossy skin and the symmetrical seizure of the joints favours a nervous origin. The most striking case I have seen was in a man over thirty, in which there was atrophy of the muscles of one half the body, following successive involvement of the ankle, knee and hip of the same side. Finally, the vertebral joints were involved. A toxin attacking the joints and nervous system may explain the condition—just as the toxin of rheumatism attacks alike the knee and the sciatic nerve.

There is no medicinal treatment which does any good except tr. ferri perchlor. A second case which received lasting benefit by treatment took an odious mixture of tr. ferri perchlor. and quinine. Potassium iodide (which is used in far too many conditions) should not be used alone, as the disease is one of depression. Tr. ferri perchlor, milk, cod oil, and stimulating food are strongly indicated.

In conclusion the chief points of my paper are (1) the clinical evidence pointing to throat invasion as the jumping-off point in rheumatism, and (2) the striking facial appearance characteristic of gout, rheumatism and rheumatoid arthritis.

July 21st, 1913.

Examination of the Blood in the Diagnosis of Lead Poisoning.—Schoenfeld has examined 556 cases with actual or suspected lead poisoning. He found no line on the gums in 44 out of 126 cases of unmistakable lead poisoning and it was distinct in only 48. It was never found in persons without teeth. The hæmoglobin averaged 62 per cent. in women and 86 per cent. in men, but the most characteristic finding was the basophile granulation in the red corpuscles and its change in tint under the action of certain stains. This may occur in other conditions, but in none can there be any confusion with lead poisoning. The workmen most exposed to the danger of lead poisoning—according to his experience—were (1) those in the powder rooms of lithographing works, (2) type founders, (3) dyers, (4) painters, (5) employees in plating works, (6) file-grinders, stereotypers, etc., and, eighth on the list, type-setters. Type-setters know the dangers to which they are exposed and apply to a physician at the first sign of trouble. Examination of the blood is peculiarly useful in these cases, as when it shows normal conditions the patient can be reassured that his symptoms are not due to lead poisoning. The blood findings are also useful to control the progress toward recovery; in eighteen of forty patients thus kept under supervision the blood returned to normal after they had changed to some other occupation. The blood findings show whether patients are under the influence of plumbism or not, and permit appropriate treatment without loss of an hour.—*Journ. A.M.A.*, vol. lxi, p. 72.

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ARTERIO-SCLEROSIS.*

By WILLIAM E. HUME, M.A., M.D.Cantab., M.R.C.P.Lond.,

Professor of Pharmacology, University of Durham College of Medicine; Hon. Physician to the Royal Victoria Infirmary, Newcastle-upon-Tyne.

THE title of this address—arterio-sclerosis—was given to me with the request that I would tell you about its symptomatology and treatment. The subject is a very large one, as arterio-sclerosis is by no means a simple clinical entity—it is a pathological result.

It may be the result of many diseases, such as lead-poisoning, diabetes, chronic Bright's disease, and many metabolic disturbances, each disease giving a different setting to the clinical picture. Even pathologically, there is considerable difference of opinion as to the uniformity of the underlying morbid changes which lead to a thickening of the arteries.

This afternoon I wish to limit your attention to the disease as it affects the smaller arteries, and try to unravel the causation of arterial thickening and its results upon some of the systems of the body.

It is necessary to say a few words about the different pathological varieties of arterio-sclerosis. Arterio-sclerosis affecting larger vessels, for instance, the aorta and iliac arteries, is a more localised, nodular affection—the so-called nodular arterio-sclerosis—and its presence cannot usually be detected in life; whereas thickening of the smaller arteries—diffuse arterio-sclerosis—produces starvation effects upon the tissues supplied by the affected vessels, and the obstruction makes it more difficult for the heart to pump the blood through them to the capillaries.

Let us briefly consider the pathological changes

* Read before the Newcastle-upon-Tyne Division of the British Medical Association.

which underlie the gradual thickening of arteries, such as can be felt in the radial arteries (diffuse arterio-sclerosis).

Jores teaches us that the first alteration is a splitting up and a proliferation of the internal elastic lamina—a subendothelial proliferation. In the neighbourhood of this increase of tissue there is fatty degeneration of the deeper cells of the intima, and the intima itself may become transformed into fibrous tissue and notably thickened. A hyaline degeneration and fibrosis takes place in the tunica media.

These changes, which lead to a palpable thickening of the radial artery, take place also in the smaller vessels right down to the capillaries, and diminish the supply of blood to the capillary field.

We must now consider the causes of this morbid change in the vessels, and I think the best classification comes from the writings of Clifford Allbutt, who has done so much to elucidate the problems of the cardio-vascular system. His classification has the great advantage of being founded upon a ripe clinical experience.

He divides cases of arterio-sclerosis into three groups:

(1) Involutionary, or decrescent.

(2) Toxic.

(3) Hyperpietic.

(1) The first group, the involutionary, is revealed in the so-called pipe-system artery. A working-man, who has hitherto been healthy, at the age of sixty to seventy years has a bad attack of bronchitis, and in the course of examination the radial artery feels as though it were made up of a series of hard rings. He has emphysema and is short of breath. The heart reveals no marked hypertrophy, and there is no evidence of renal disease.

In such a case we are usually surprised to find that the blood-pressure is not raised, though the absence of cardiac hypertrophy should keep us right and negative this surmise. In this type the change is chiefly in the middle coat—firstly degeneration of the muscle-fibre, and then calcification.

If the brachial arteries are in a condition similar to that found in the radial arteries, it may require a greater force to obliterate the vessel while using the sphygmomanometer, as the extra force is spent in squeezing the calcareous arteries.

In all cases of this type in which the brachial arteries could not be distinctly felt, I have never discovered a permanent increase of blood-pressure, such as occurs in arterio-capillary fibrosis.

There is one vascular complication to which individuals with this type of artery are liable, and that is gangrene. When the middle coat is calcified in this way, a pit occurs inside the lumen of the vessel, and an ante-mortem clot is liable to form in it; if the clot is dislodged, a terminal vessel is blocked; gangrene results, because the neighbouring vessels are so ruined that a compensatory circulation cannot be formed.

This group, then, is characterised by calcification in the middle coat of the arteries, absence of hypertension and cardiac hypertrophy, and a liability to gangrene.

(2) Of the toxic group, due to lead, diabetes, and so forth, I do not intend to speak. Suffice it to say that the changes in the vessels are merely an incidence in a more general disease, and each disease reveals its identity by its own characteristics. The secondary changes in the cardio-vascular system, such as hypertrophy of the left ventricle and increase of blood-pressure, will be discussed when the third group—the hyperpietic—is under discussion, as the results of both types are the same.

(3) The third group demands more particular attention, for the following reasons. First of all, it has been more recently separated from the motley group of arterio-sclerosis, and also its early recognition is imperative for its proper treatment.

Up to the 'eighties it was held that arterio-sclerosis, in whatever way produced, always resulted in a gradual rise of blood-pressure, and that the hypertension was invariably dependent upon the thickened arteries.

At this time Clifford Allbutt and Huchard independently pointed out that an increase of blood-pressure did not always give rise to arterio-sclerosis; and, further, Clifford Allbutt showed that a high blood-pressure might exist without any change in the vessels at all. This condition he called hyperpiesis. It is now held that this increased blood-pressure, unchecked and persistent, may lead to the pathological condition of the arteries which we have described as arterio-sclerosis. The word "hyperpiesis" merely means increased pressure, and we must consider the factors which cause it, and the effects which it produces.

May I illustrate the condition by quoting to you a case which will recall the type?

A man in good position, æt. 50 years, was sent to me last October. He was a big, strong man, who had always led an athletic life. In spite of having treated himself too indulgently to alcohol and tobacco, he had never had a day's illness. He complained that recently, while going up a slight hill to the station, he had been seized with pain in the chest, which had occasionally made him stand still for a moment or two.

On examination, I found that his radial artery was full and difficult to compress, though the wall itself was not thickened when the vessel was emptied. The blood-pressure was 200 mm. of mercury. The left ventricle was distinctly hypertrophied, and the heart impulse was forcible, the apex-beat being displaced downwards and outwards.

The urine was of moderate specific gravity, and there was no albumen.

Where was the fault in this case? Certainly not in the kidneys, as high blood-pressure which follows kidney disease is always associated with a thickening of the vessel-wall. Also there was a reasonably high specific gravity, and no albumen, and there was an absence of casts.

One cannot lay the fault to the heart. An essential hypertrophy may occur, but if the arterio-capillary system is normal, the hypertrophy is not associated with an increase of blood-pressure.

Recognisable changes were certainly not present in the arteries.

We are reduced, then, to regard the hypertrophy of the left ventricle as secondary to the increase of blood-pressure. What, then, may be the factor which determines this increase of blood-pressure? There are only two ways, as far as I know, of looking at the matter. Either the blood, in some way or other, becomes physically altered so that the friction is increased between the blood and the vessel-wall—such has been referred to as increased viscosity of the blood—or there is some narrowing of the arterio-capillary field, such as might be caused by spasm of the smaller arterioles. Increased viscosity is extremely difficult to demonstrate. Many experiments have been made of running blood through tubes of fine bore, and comparing the times taken for blood from this case and similar cases to flow through them. By such means it has been quite impossible to demonstrate that blood from cases of so-called hyperpiesis takes longer to flow through the tube than normal blood.

Most people have given up the idea that such a thing as increased viscocity of the blood exists at all. We are left, then, with some narrowing of either the lumen of the smaller vessels or some restriction of area in the capillary field.

Osler states that the fault lies between the capillary cells and the lymph-spaces. He suggests that the stoppage occurs in the so-called working area of the body, where the exchange takes place between the substances brought by the blood to the cells, and where the waste products are given up by the cells to the blood. Others consider that the essential difficulty lies in spasm of the muscle-coats of the smaller arterioles, which diminishes their lumen, and in this way an obstruction to the blood-flow is caused. Much experimental work has been done to prove this condition of so-called hypertonus or spasm, and as we shall see, the theory meets with considerable clinical support. Though the existence of the condition is admitted by all, as yet we are quite ignorant of the causes which lead to this spasm of arterioles. Or, according to Osler's view, we are unaware of the conditions which cause the stagnation in the smallest capillaries. As patients suffering from hyperpiesis are frequently subject to bilious attacks and other digestive disturbances, and as they are cured by treatment which improves elimination, some disorder of metabolism is said to be the essential cause. Such an assertion is mere verbiage, and does not bring us nearer to the real truth of the matter. In this connection, however, it is an interesting fact that D'Amato produced increase of blood-pressure, and, finally, arterio-sclerosis, by feeding dogs on putrid flesh.

We must now consider the results to the patient of this condition of hyperpiesis, or essential increase of blood-pressure.

Headache, drowsiness, morning fatigue, ineptitude for work, cold extremities, tinnitus, giddiness and attacks of migraine are some of the minor ailments attributed to hyperpiesis.

Clifford Allbutt lays considerable stress on the fact that patients with this disease sleep uneasily and wake unrefreshed, and that they are particularly susceptible to the effects of alcohol and tobacco.

We owe to Russell many excellent descriptions of the effects on the central nervous system of temporary spasm in the cerebral arteries.

I have recently had in the hospital a case which exemplifies this condition very well.

A man, æt. 40 years, with a high blood-pressure and a hypertrophied left ventricle, suffered for a day or two from headache and mental confusion. He became more or less suddenly paralysed in the right arm and leg. This condition passed off in less than a week, and he is now perfectly well. Other patients become temporarily aphasic—and sometimes aphasia and hemiparesis exist in the same patient, where the spasm has involved the middle cerebral artery of the left side of the brain.

A farmer, æt. 64 years, who had a high blood-pressure for many years with no evidence of renal disease, woke up one morning to find that his leg felt dead. He tried to move it, but was unable to do so. He knocked through the wall to the next room, and summoned the nurse who was attending his sick wife. When she reached him he was aphasic, and unable to describe what was the matter with him, and he was completely paralysed on the right side of the body. This was at 6 a.m. The doctor, who lived six miles away, was urgently sent for, and when he arrived, at 9 o'clock, the patient was sitting up in bed eating his breakfast and reading his newspaper, perfectly recovered.

One can only believe that such temporary conditions are caused by spasm of vessels, producing a temporary ischæmia of the brain. By the same temporary spasm, pseudo-gastralgic attacks may be produced, with deep-seated pain below the umbilicus lasting one hour or more, and sometimes accompanied by vomiting and distension.

Temporary spasm in the crural arteries gives rise to the condition in the legs known as intermittent claudication.

Before we consider the outlook and treatment of hyperpiesis, it is necessary to see what the result will be if the condition goes untreated. There is no doubt that high blood-pressure will, in the end, produce degenerative changes in the vessels. If the aorta is compressed, arterio-sclerosis occurs on the side nearest the heart. If adrenalin and pituitary extract—substances which raise blood-pressure—are injected into animals, changes are produced in the arteries which have all the characteristics of arterio-sclerosis. So also, clinically, cases have now been followed by Clifford Allbutt, Russell, and many others, who, having suffered from high blood-pressure without demonstrable changes in the vessel-walls, eventually developed thickened arteries.

In this—what we may call a second—stage of the condition, when secondary changes have occurred in the vessel, the physical signs reveal thickening of the radial artery, which, emptied of blood, can be rolled under the finger, a forcible apex-beat in the fifth or sixth interspace in, or outside of the nipple-line, and a heightened blood-pressure. It is obvious that other vessels beside the radials will be affected, and the symptomatology and the course of the disease will depend upon the distribution and degree of the diseased channels.

In this way the types of the disease fall into cardiac, cerebral, renal, etc., types. There is only time at my disposal, however, to mention some points in connection with each of these types.

In many cases the heart fails behind the excessive load which it has to lift at each beat. Shortness of breath, anginal attacks, and all the manifestations of gradual heart-failure are very common clinical pictures. The local disease in the cerebral vessels may, under the influence of constant or intermittent high blood-pressure, result in cerebral hæmorrhage.

It seems certain that the kidney-vessels at times bear the brunt of the disease, and chronic interstitial nephritis results, and the first evidence of gross disease of the kidneys may be revealed by one of the manifestations of acute uræmia.

Even after the vessels have become grossly thickened, many of the temporary nervous disturbances—aphasia, hemiparesis, etc.—which have been discussed under hyperpiesis, may still occur from time to time, from superadded spasm of the vessels.

Before leaving this stage of the disease I would like to mention the fact that many cases of atrophic emphysema of the lungs are probably caused by arterio-sclerotic changes in the pulmonary arterioles.

Knowing that nothing can release the artery walls from its bonds of fibrous tissue, it is not surprising that the majority of patients with arterio-sclerosis eventually die of heart-failure. Though some few at an early age are carried off by apoplexy,

and others develop chronic interstitial nephritis, the large majority become more and more short of breath, the legs become œdematous, and a loud, blowing, systolic murmur is heard at the apex. Such cases form the picture of the so-called "heart and kidney case."

With regard to treatment, it will be convenient to consider it under the three headings into which we have grouped our cases.

In the third group, or hyperpiesis, in which we have concluded that there is simply high blood-pressure, dependent upon obstruction in the neighbourhood of the capillary field, our object ought to be to so alter the metabolism of the body that elimination becomes more perfect.

Alcohol and tobacco must be cut down to a minimum, and the diet should be of the lightest, with especial avoidance of the red meats. A moderate amount of exercise is essential, and cold baths ought to be given up for a time, though a warm bath in the morning is advocated by all. As you know, cold applied to the skin materially raises blood-pressure.

As to drugs, those which are directed towards the prevention of fermentation and putrefaction in the intestines are essential, and of these calomel—gr. ij or iij. twice a week—is the most effective with a morning saline aperient. Vaso-dilators have not the power in the human body in therapeutic doses that they have in the animal body in the laboratory, and I have frequently tried to lower blood-pressure permanently with various members of the nitrites without success.

Further, it is well known that individuals with thickened arteries and high blood-pressure are better in every way when the pressure is moderately high than when, by much purging and low diet, it has been reduced 20 to 30 per cent.

There is no doubt that when the dietetic and hygienic improvements can be made as ideal as possible, hyperpiesis pure and simple can be diminished or eradicated.

The difficulty is to get individuals of this type to submit to any form of restraint. The patient resents any radical alteration in his manner of life. Emmerson says: "Men like your patients, full of coarse strength, butcher's meat, and sound sleep, will suspect any poetic insinuation, or any hint for the conduct of his life, which reflects upon this animal existence."

When the arteries have become permanently thickened the above line of treatment is still necessary, but we have now reached a stage when the condition of the heart usually causes anxiety.

As I have said, hygiene, diet and elimination are the first essentials, and, when the heart fails, the necessary amount of rest.

Those hearts which have failed, and have developed auricular fibrillation, detected by complete irregularity of the pulse, frequently do very well with large doses of digitalis.

The heart which fails with a regular pulse reacts best to strychnine and caffeine.

Besides these methods of general treatment, certain measures must be taken to meet certain emergencies, and I will conclude by drawing your attention to the advantage of venesection.

The removal of 16 to 20 oz. of blood from a vein at the front of the elbow-joint frequently brings very great relief, especially in individuals of the plethoric type.

In angina pectoris, which is a paroxysm of vaso-motor spasm, amyl nitrite must be administered by inhalation. As I have already stated, the other nitrites are mostly quite ineffective.

Lastly, there is no doubt that small doses of potassium and sodium iodide, continued over a long period, are beneficial in case of arterio-sclerosis with high blood-pressure. Though we are quite ignorant of the mode of action of the iodides in this state, there is no doubt of their occasional therapeutic advantage.

In this brief survey of a very large subject I have only endeavoured to separate certain types of the disease, in order that the recognition of these types may enable you to classify and so treat your cases more clearly.

July 28th, 1913.

Radiotherapy in Gynæcology, especially for Carcinoma of the Uterus.—Döderlein reports 32 cases of myoma and hæmorrhage treated by radiotherapy with prompt and permanent benefit and without any bad effects. The tumours entirely disappeared in many cases. More surprising, he found a combination of Röntgen ray and mesothorium treatment effective in many cases of uterine cancer. He thinks the rays had a direct effect on the carcinoma-cells, and not, as has been previously held, that the cancer-cells were affected secondarily by contraction of the surrounding connective tissue. —*Journ. A.M.A.*, vol. lxi, p. 72.

SOME INTERESTING SURGICAL CASES.*

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GENTLEMEN,—It has been said, aptly enough, that "He who makes no mistakes makes nothing," and as surgery is far from being an exact science our mistakes are by no means infrequent. We have always before us that great saying of Hippocrates that we should be careful not to harm our patients even when we are striving to help them. I have thought, therefore, that I might profitably employ these two clinical lectures by calling your attention to some of the mistakes into which we have lately fallen in the hope that by hearing about them you might be prevented from similar errors.

(A) FOREIGN BODY IN THE ŒSOPHAGUS.

(1) The first case I shall speak to you about is that of a foreign body impacted in the œsophagus. It would seem impossible in these days of X-ray photographs and bronchoscopes that a foreign body should remain impacted in the œsophagus for two or three days and that we should then make a series of mistakes in our attempts to remove it. But here is a case—for this married woman, æt. 32 years, was admitted to Lucas ward on Thursday morning, April 17th, saying that she had swallowed this tooth-plate, which you see is made of vulcanite and has one incisor tooth attached to it. She had no difficulty in swallowing, and, as the X-ray department was closed and the screen does not appear to have been used, she was allowed to go home and was told to come again on Friday morning. She did not return until the following Monday, when she said that she could only swallow liquids and even they caused pain. An examination of the throat by means of the œsophagoscope showed that the plate was embedded in the wall of the œsophagus at a point ten inches from the teeth. The mucous membrane was œdematous and it proved to be impossible to remove the plate by cutting or breaking it. The patient was therefore anæsthetised with ether administered by the intubation method and a bougie was passed down the œsophagus until it entered the stomach. No resistance was encountered, and this was explained by the fact that the œdematous mucous membrane had "pocketed" the plate and thus prevented the bougie from touching it. The bougie was therefore withdrawn until only ten inches of it remained in the gullet. An incision was then made along the posterior border of the left sterno-mastoid muscle, and the point of the bougie being pressed against the inner wall the œsophagus was made to project into the wound. This movement also flattened the common carotid artery and stopped its pulsation, so that when the wall of the œsophagus was incised the artery was wounded. A double ligature was applied above and below the puncture and the opening in the œsophagus was enlarged until a finger could be introduced. A slit in the mucous membrane marking the place where the tooth-plate had been impacted was easily felt, but the plate itself could not be located, and the bougie, which had been withdrawn through the mouth, was again passed into the stomach through the wound without any hindrance. It was assumed, therefore, that the foreign body had either entered into the stomach or had passed through the wall of the œsophagus into the mediastinum. The latter position was thought to be very unlikely, as the pulse and respiration of the patient remained unchanged. A rubber drainage-tube was put into the œsophagus, which was closed by a few point sutures in the greater part of its extent and the superficial wound was closed in a similar manner, the tube being secured to the edge of the skin by a silk suture. The patient made so good a recovery that on the following day she was taken to the X-ray department, when the plate was found to be lying in the stomach. I thought that it might be necessary to remove it by a gastrotomy, but fortunately determined to wait for a few days to see what Nature would do, and on the following Thursday—just a week after it had been swallowed—the plate was passed by the bowel whilst the patient was at stool. The tube was left in the œsophagus for forty-eight hours and the wound in the neck healed rapidly by granulation. You will see that she has made a safe recovery, in spite of the fact that on the Monday after she had passed the plate, and exactly a week after the operation, she aborted of a four months' fœtus.

* A Clinical Lecture delivered at St. Bartholomew's Hospital.

Now here was a series of mistakes. The patient ought to have been more careful about the condition of her tooth-plate—the initial mistake. When she came to the hospital she ought not to have been sent away until we had satisfied ourselves that the tooth-plate was not in her œsophagus, and when she had been sent away she ought to have returned when she was told to do so, and not have waited until the pain and difficulty of swallowing compelled her to come back. The failure of the œsophagoscope demonstrated how difficult it is to deal with vulcanite plates when they are firmly wedged low down in the gullet; but when it had failed we ought not to have assumed that the plate would remain where it was. When she was under an anæsthetic, and the passage of the bougie failed to reveal the presence of a foreign body, we ought not to have assumed that it was still there. It would have been better to wait until a skiagram could be taken rather than subject the patient to the risks of an operation which proved unnecessary. During the operation I ought not to have punctured the common carotid artery. I knew perfectly well where it lay, as I taught you operative surgery for thirteen years, and purposely made my incision into the wall of the œsophagus as far back as possible, but I did not realise sufficiently that the pressure of the bougie would flatten it out to such an extent as it did.

Looking back and hearing all this in cold blood the mistakes seem obvious and easily avoidable, but in a similar case you may easily fall into any or all of them. The initial mistake you will never be able to prevent, because as long as people use artificial teeth so long will they continue to be careless about the condition of the plate. In many cases a plate which is swallowed is passed readily enough *per vias naturales*, but in a certain proportion it becomes impacted at some point in the œsophagus, and what is true of a plate is equally true of other foreign bodies, such as coins, darts, and even marbles. When a foreign body has become fixed below the level of the cricoid cartilage it is surprising how little discomfort it may cause. It produces some œdema of the mucous membrane and perhaps a local paralysis of the muscular coats of the œsophagus. This paralysis allows the foreign body to lie in a pouch or hernia of the wall, where it is undiscoverable by any bougie or other exploratory instrument, and here it may set up a process of ulceration, which leads to perforation of the œsophagus and kills by suppurative inflammation of the mediastinum or by broncho-pneumonia. Here is such a case, with a most unlikely cause—a marble.

(2) The child was four years old, and she swallowed the marble on July 30th, 1888. She was brought to the hospital on the following day, and her mother stated that she could not keep her food down. "She did not vomit but it just came back." She was put under chloroform, and a No. 10 gum-elastic bougie was passed. A slight obstruction was felt at the cardia, but this gave way when a little pressure was exerted, and the bougie readily entered the stomach. The child was sent home, and was brought back again by her mother on the following day—August 1st—who said that the child was no better. She was sent home with advice as to what might be done, but she returned on the next day—August 2nd—and was then so ill that she was at once admitted. Mr. Savory passed a bougie, and found that there was some obstruction at the lower part of the œsophagus, but the instrument could be made to clear its way and enter the stomach. The child died at 1.45 a.m. on August 3rd, four days after the marble had been swallowed.

The specimen, which is preserved in the museum (No. 1867*b*), shows that the œsophagus is ulcerated for about an inch immediately above the cardiac orifice. It is so much softened that its wall gave way as soon as it was removed from the body, leaving a large and ragged opening through which the small black marble escaped.

These two cases will show you how troublesome a condition may arise as a result of the impaction of a foreign body in the gullet. In the first case an operation might have been avoided; in the second case it was imperatively demanded, but it was the recollection of this latter case which caused me to operate in the first one.

(B) Pneumonia simulating Abdominal Inflammation.

It is well known that an acute onset of pneumonia may simulate acute abdominal inflammation. Mistakes have been made where acute appendicitis has been thought to be pneumonia, and where the perforation of a duodenal ulcer has been looked

upon as pneumonia. Here is a case where acute pneumonia was thought to be a ruptured liver.

The patient was a vanguard, æt. 16 years, who was admitted into Henry ward unconscious on January 21st, 1913.

He was brought to the hospital at 6 a.m. in an ambulance, as he had been found lying by the side of a railway line in a goods yard where shunting was taking place. No history of an accident could be obtained, but when the patient recovered consciousness about two hours later he complained of great pain over the lower ribs on the right side and he brought up some blood. On the arrival of his friends we learnt that he appeared to have been in perfect health on the previous night when he went to bed, and that he had started off for work as usual at 5 a.m. on a raw January morning.

Examination showed that he had a temperature of 99° F., pulse 96, and respirations 44. The urine had a specific gravity of 1024; it did not contain any albumen, sugar or blood.

The thorax moved well on respiration, but there was less movement on the right than on the left side. The heart-sounds were natural, and the lungs were apparently normal. The pain extended indefinitely over the right side of the chest in front of the mid-axillary line. No crepitus could be felt or heard over any of the ribs on the right side.

The abdomen moved well on respiration, but there was great tenderness and rigidity in the right iliac fossa. There was no fracture of the pelvic bones, and the abdomen was hyper-resonant over the rigid area.

When I saw the patient at 3 p.m. he was more collapsed that he had been in the morning. There was marked movement of the alæ nasi during respiration. The abdomen was a little distended in the lower part, and was rigid on the right side. The pulse was 120, respirations 36, and temperature 100° F. I thought the patient had sustained a slight rupture of the liver with some bruising of the lower lobe of his right lung, and that he was probably bleeding into his peritoneal cavity. I told Mr. Neve—my house-surgeon—that I was prepared to open his abdomen if the symptoms became worse. The patient slept badly, and on the following day he was found to be suffering definite pain in the right side of his chest, the abdominal symptoms having all passed off. There was dulness in the right axilla, and there was a patch of dulness, over which bronchial breathing could be heard at the right base. A diagnosis of pneumonia of the right lower lobe was made, and this was confirmed by my colleague, Dr. Ormerod, who had the patient transferred to one of his own wards, where he made a speedy recovery.

The disturbing factors in this case seemed to me to have been the sudden onset, the unconsciousness, the vicinity of the railway and the reference of the symptoms to the right side of the abdomen rather than to the lung. We erred because we failed to attach sufficient importance to the blood which was brought up, to the working of the alæ nasi, to the rapid respiration, and to the rising temperature. It was only when the abdominal symptoms diminished and the chest signs increased that we arrived at a correct diagnosis.

(c) Acute Duodenal Perforation simulating Pneumonia.

In the next case an exactly contrary mistake was made, for a case of acute duodenal perforation was looked upon as a case of pneumonia.

A porter, æt. 41 years, was admitted to the hospital on May 23rd, 1902, saying that he was at work and quite well until eleven a.m., when he was suddenly seized with pain in his epigastrium. The pain continued and he vomited several times before coming to the hospital. He had passed no flatus since the pain began; his bowels were well opened on the previous day. He was a temperate man and there was no history of indigestion. When he was seen at 2 p.m. he was reported to be a well-nourished man in obvious pain. His tongue was clean and moist; his respirations were very shallow and 60 a minute; his pulse was of fair volume and tension, regular, and 100 a minute. In the chest the percussion-note was impaired at the right base, and the entry of air at the right base was weaker than at the left. There were no additional sounds. The pain in the abdomen was not localised, but the patient complained of it chiefly over the upper half and down the right side.

The abdomen was not distended, but it moved very little during respiration. The movement, however, was equal all over, though the abdomen was held somewhat rigid. It was everywhere

tender and tympanitic, except that the liver dulness was present. Nothing abnormal could be felt. It seemed impossible at this time to make any definite diagnosis, and directions were left that the patient should be carefully watched on the assumption that the case was rather one of pulmonary than of abdominal mischief.

There was no definite change at 6 p.m. An enema saponis had been retained, but the pain was relieved by poulticing. The temperature at this time was 99·8° F., the pulse was 120 and the respirations 60. At 12 o'clock, midnight, there was still no material change in the condition of the patient, whose temperature was 99·8° F., pulse 120, respirations 60–70. A little liquid had been vomited. At 1 a.m. the abdomen was distended and the patient was slightly collapsed with a pulse of 144, almost running. At 2 a.m. the patient was still more collapsed, and I then saw him for the first time. He appeared to be so ill and was so obviously suffering from peritonitis that I determined to open his abdomen without farther delay. As soon as I did so gas and fluid escaped from the wound, and when the stomach was drawn up an ulcer was found upon its posterior surface close to the pylorus with an aperture large enough to admit a full-sized probe. There was a considerable deposit of plastic lymph in the neighbourhood of the perforation. An attempt was made to close the hole by means of Lembert's sutures, but the operation was rendered difficult partly by the awkward position of the ulcer and partly by the rotten state of the tissues, which allowed the sutures to cut out repeatedly. Four sutures were passed, and it appeared that the opening had been successfully closed since no more fluid poured out through the ulcer.

The patient bore the operation badly and his pulse was almost imperceptible at the end of it. At 4 a.m. he was transfused, but at 7.30 a.m. he died. A post-mortem examination made at 3 p.m. the same day showed that the whole peritoneum was acutely inflamed, its endothelial aspect being covered with a layer of fibrino-purulent lymph. There were collections of pus at the bottom of Douglas's pouch, in the lumbar region and in the right subphrenic space, this space being almost completely shut off from the rest of the peritoneal cavity. There was a conical ulcer measuring half an inch in diameter at the upper and back part of the first portion of the duodenum. It had sharply cut edges, but there was no infiltration of the margin. The floor of the ulcer had perforated, but the sutures were not accurately applied as water and intestinal contents easily passed through.

The particular interest of this case lay in the fact that the localised peritonitis must have lasted a much longer time than the onset of the symptoms would have led one to suppose; whilst the symptoms when they appeared were so obscure as to make it seem that the patient was suffering from pneumonia rather than from peritonitis. These symptoms were due undoubtedly to the subphrenic abscess.

(D) Multiple Infection of the Tongue and Glands.

A man, aged 54 years, was admitted into Henry ward on March 12th, 1912, on account of a chronic ulceration of the tongue, which was partly syphilitic and partly epitheliomatous. The patient was advised to have his tongue removed but refused to consent, saying that he would be ruined if he were unable to speak plainly. He was therefore given an injection of salvarsan and was shown how to paint his tongue with a solution of chromic acid of the strength of ten grains to the ounce. Considerable improvement resulted but the epitheliomatous ulceration remained. The patient returned to his home. A month later he came back to the hospital saying that his tongue was now more fixed and had become painful, the pain running up the side of the face. He was willing to undergo an operation. The whole tongue was accordingly removed by Whitehead's method after a preliminary laryngotomy had been performed. A section was taken from the ulcerated portion and was shown to be undoubtedly epitheliomatous. The patient made a good recovery and was sent home for a short time to recruit before the affected lymphatic glands were removed. He returned to the hospital in May with the whole of the neck inflamed and the cervical glands suppurating. Incisions were made into them and the pus was evacuated. Treatment by fomentations soon led to comparative recovery, but it was not until August that it was thought wise to undertake any formal operation for their removal. The left submaxillary salivary gland and the cervical fascia with the contained lymphatic glands were removed by Sir Henry Butlin's method,

and the tissue was sent to the pathological laboratory in the expectation that the usual report of their cancerous nature would be received in due course. When the report arrived, however, it stated that the glands were undoubtedly tuberculous and that there was no trace of carcinoma. The patient was again sent home as no enlargement of the glands on the right side of the neck could be felt. In January of this year he was readmitted to the hospital, and on January 11th the submaxillary salivary gland and some enlarged lymphatic glands were removed from the right side of his neck. A microscopical section was made, and it was reported that there was no evidence of malignant disease nor of tubercle. It was also noted that the "mouth was free from ulceration and presents a healthy appearance."

The patient was not seen again until April, 1913, when there was some ulceration of the right fauces and there was a large sloughing epitheliomatous ulcer on the right side of the neck. He complained much of pain and had become very emaciated since he was last seen in January. During the last month he has gone rapidly from bad to worse. I hoped to have shown him to you to-day, but I hear that he is too ill to travel.

The case is interesting for several reasons. In the first place because the association of tubercle and cancer is of the rarest accurrence. Syphilis and cancer of the tongue is usual, and, I believe, the rule. The ordinary history of epithelioma of the tongue is syphilis twenty or thirty years previously—this man had a primary sore twenty-six years ago—heavy smoking and neglected teeth. But cancer associated with tubercle is so rare that Sir James Paget in one of his latest lectures declared that they were relatively incompatible diseases, and you remember that throughout a long life he had devoted special attention to the history and causation of cancer, and that he had unrivalled opportunities of studying the inter-relation of diseases both in public and in private practice.

(E) Recurrent Spontaneous Hæmorrhage into the Breast. Hæmophilia?

Hæmophilia is so excessively rare in the female that Dr. Drysdale, in an article in the recently published book on the 'Diseases of Children,' goes so far as to say that no well-authenticated case of hæmophilia in a woman has been recorded hitherto. The following case, therefore, is of extreme interest, for if it must not be labelled hæmophilia because no ancestral history of bleeding is attainable, it is certain that the patient had recurrent spontaneous hæmorrhage, and as hæmophilia must begin somewhere and at some time it will be worth while in the future to trace the history of her children. The facts are as follows:

Daisy Berry, æt. 25 years, married, was admitted into Paget Ward on April 16th, 1909, complaining of a painful swelling in the left breast. She first noticed a swelling and bruising in the upper part of her left breast at the end of March. The swelling occurred quite spontaneously and she was certain that there had been no injury. It was thought to be an abscess and was incised. No pus was found but it bled excessively, and although the incision healed well the whole breast became bruised and painful. She states that the right breast was very painful and of a dark colour in the summer of 1908 in consequence of a slight injury. At the age of nine years she had a spontaneous swelling of the right ankle which afterwards "burst" and discharged "black blood." She has repeated attacks of nose-bleeding which occur once a week and may soak as many as six handkerchiefs daily. After one of these attacks, which may last several weeks, she remains free for a considerable time.

She bleeds for about an hour after extraction of a tooth, and she reports of her menstruation that it is irregular and scanty—once in about seven or eight weeks.

The patient is a healthy looking but pale young woman who blushes easily and yields a well-marked *tâche* on the skin of her chest. She has two healthy boys, and no family history of hæmophilia is obtainable.

Examination of the blood shows that it has a coagulation-time of ten and a half minutes, the control—from an apparently healthy dresser—being three to six minutes. The leucocytes are 9800, the red blood-corpuscles 4,900,000. Five c.c. of blood taken from the left median basilic vein were put into broth and agar tubes. No growth was obtained in any of them.

Whilst the patient was in the hospital she had severe pain in her breast and some spontaneous hæmorrhages occurred. From May 5th to 11th her temperature ran up to 103·8° F. without any

obvious cause and afterwards returned to normal gradually. She left the hospital on May 15th and was readmitted on July 22nd, 1909, saying that the pain and bleeding in her breast had become worse. Examination showed a blackened area of skin below the nipple and there were several nodules in the glandular tissue of the breast. The largest of these nodules had definite edges and floated in the breast-tissue like a fibro-adenoma.

The patient was kept under careful observation in the hospital until August 3rd so that all suspicion might be excluded that the bruising was self-inflicted. During this interval the bleeding seemed to be continuous as the whole breast became black with extravasated blood. She also suffered severe and continuous pain, and her temperature ranged from 99° to 101° F.

The breast was removed at her own urgent request on August 3rd, when the temperature immediately fell. The wound healed by first intention so that she left the hospital on August 17th.

Subsequent examination showed that there were no enlarged axillary lymphatic glands. The section, which is preserved in the Museum (No. 3142 g.), is described in the catalogue as "Part of a female breast showing spontaneous extravasation of blood into the capsule of the mammary gland. The mammary tissue itself appears normal, but is surrounded by a hæmorrhagic zone. On one side there is a slight infiltration of the escaped blood into the superficial parts of the gland. The skin covering the affected area has a bruised appearance. Microscopic examination shows that the extravasated blood lies among the fibres of the capsule."

The patient left the hospital in August, 1909, and as I had heard nothing about her afterwards I wrote to Mr. W. G. Loughborough, under whose care she was, asking for further information as to her progress. He wrote on April 21st, 1913, "*Re* Mrs. Daisy Berry I have not seen her for about a year, but when she came to see me then she was perfectly well and had had no return of her old trouble. In the previous autumn (*i.e.* 1911) she had a healthy child after a normal labour. The child died of broncho-pneumonia at the age of five months."

(F) Spontaneous Fracture of the Thigh Secondary to Cancer of the Uterus.

Secondary deposits in bone are not uncommon in cancer of the breast. They are usually in the humerus or in a rib, and they lead to spontaneous fracture. I do not remember to have seen a case where cancer of the uterus was associated with spontaneous fracture of the femur until the following case came under my observation a few weeks ago:

An unmarried woman, æt. 50 years, was admitted to Lucas ward on April 1st, 1913, complaining that she had lost the use of her right leg. She stated that she had been in good health until December, 1912, when she had "a bad cold" followed by "rheumatic pains" in the legs, worse on the right than on the left side. The pain in the right leg continued until she was unable to walk upon it any longer, and she finally took to her bed five weeks before she was admitted to the hospital. The pain was severe on movement, but it was not worse at night, and it never travelled below the knee. After she had been in bed for a month the thigh became deformed, and the pain then diminished greatly. She became more helpless, and, for want of proper nursing, bed-sores began to be formed.

Thirteen years ago an ovarian cyst was removed in the hospital, and a reference to her notes show that both ovaries were removed for cystic disease. They were examined microscopically but showed no signs of malignancy. On her admission to the hospital on this second occasion her heart and lungs were found to be natural. In the abdomen the lower edge of the liver was not palpable; there were no enlarged glands in either groin, and the lumbar glands could not be felt. The abdominal muscles were held rigid, but an intra-abdominal tumour could be distinguished in the right iliac fossa. It was sausage-shaped, hard, and fixed to the surrounding structures. It had a rounded upper border and rounded edges. The lower border was indefinable. It was dull on percussion.

The patient lay partly upon her left side with the right leg flexed at the knee and thigh. The whole leg was markedly abducted and was internally rotated, more so than was normally possible. The leg was œdematous. At the knee flexion was natural, extension was incomplete; there was no fluid in the joint.

At the right hip abduction and external rotation were very limited, flexion and extension were incomplete. All power of voluntary movement was lost. There was a point of maximum tenderness over the great trochanter. Sensation was

unimpaired. There was no alteration in the relation of structures as measured by Nélaton's line, and the measurements of Bryant's ilio-femoral triangle were identical on the two sides. The right leg was two inches shorter than the left. A vaginal examination, made by Dr. Williamson, showed that the whole pelvis was infiltrated with malignant growth, and the cervix uteri was the site of a mass of friable and easily bleeding cancer, which was fungating. A skiagram showed that the femur was fractured obliquely just below the great trochanter, and the whole bone in the neighbourhood of the fracture was greatly rarefied.

The patient was made as comfortable as possible by means of a weight and extension applied to the leg, and she was discharged to a home for the dying on May 7th, as it was impossible for her to obtain proper attention at home. She died there on May 25th.

July 28th, 1913.

Chronic "Non-Gonorrhœal" Urethritis.—S. H. Likes and H. Schoenrich report cases of non-gonorrhœal chronic urethritis. The discharge may be due to various causes—bacteria, prostatic conditions, rheumatism, bladder disease, syphilis, or grippe, and, most frequently, to granulations or infiltrations of the urethra. This type was first described by Otis and Overlander. The usual symptoms consist in a purulent drop collecting at the meatus from time to time, causing little pain but much anxiety in many cases. When a thorough examination has proved the absence of gonorrhœal organisms or other infections and any involvement of the posterior urethra, prostate, etc., one may be certain that the focus is in the anterior urethra. This must be found by the introduction of a bougie *à boule* of sufficient calibre to stretch the urethral wall so that the granular and infiltrated areas may be felt as rough spots or absolute obstructions. To demonstrate these lesions requires instruments of larger calibre than commonly used. The one thing likely to interfere with their detection is a too small meatus. The treatment is dilatation by bougies *à boule* of gradually increasing calibre and ironing out the rough areas by gentle massage made painless by the introduction of cocaine. Cure will result if all parts of the anterior urethra are restored to the normal calibre.—*Med. Record*, vol. lxxxiv, p. 29.

VASO-MOTOR PHENOMENA AS A PREMONITORY SYMPTOM IN RHEUMATOID ARTHRITIS.

By JAMES LINDSAY, M.D., M.R.C.P.,
Hon. Physician, Royal Mineral Water Hospital, Bath.

DURING the past six years my attention has been directed to the subject of vaso-motor disturbances as they occur in rheumatoid arthritis. Spender, a former physician to the hospital, was the first to describe those changes in connection with rheumatoid arthritis. He described the extreme pallor often followed by the deep purple tint with coincident symptoms of intense coldness and localised sweatings of the palms which obtained so commonly in rheumatoid patients, but throughout his writings he never definitely alludes to them as premonitory phenomena. The more recent observations of Bannatyne and Llewellyn have also been brought before the notice of the profession.

The observations here embodied are derived from 153 consecutive cases of true rheumatoid arthritis (as distinct from osteo-arthritis) seen in the Royal Mineral Water Hospital, Bath. No fewer than 108 exhibited vaso-motor disturbances. These ranged in degree from slight numbness and blanching of the terminal phalanges of one or several fingers to symptoms so severe as to be indistinguishable from true Raynaud's disease. Clinically there is little to distinguish vaso-motor disturbances met with in rheumatoid arthritis from the phenomena of Raynaud's disease.

These disturbances are in many instances followed by swelling and the other changes in the joints seen in the typical spindle-shaped interphalangeal joints of the fingers, but even here there is a point of resemblance, as Raynaud himself described joint changes in connection with the vaso-motor phenomena, and writes of a condition of fibrous-ankylosis of the terminal interphalangeal articulations.

These symptoms of rheumatoid arthritis are largely overlooked for two reasons: firstly in the majority of cases they are slight and transitory in nature, and not evoking pain are disregarded by the patient; secondly they generally occur some time previously—even months or years before, and from the above causes have made no lasting

impression on the minds of the patient. Patients themselves rarely mention these disturbances voluntarily, and inquiry as to the former occurrence of any "peculiar sensations" affecting the fingers will generally bring out a history of "dead fingers," "pins and needles," etc.

These disturbances are of considerable importance in connection with the premonitory symptomatology of rheumatoid arthritis; I believe that they constitute its earliest manifestations. Of the 108 cases in which vaso-motor changes were present, these symptoms appeared previous to any changes in the joints in no fewer than 103, while in two of the remainder the vaso-motor changes preceded the involvement of fresh joints, occurring in the course of an exacerbation.

The interval between these vaso-motor disturbances and the appearances of the joint swellings varies within wide limits. It may be only a few weeks; but, on the other hand, the patient may be subject to vaso-motor symptoms for many years before there is any indication of involvement of the joints.

The following is a brief account of some of the cases:

E. B—, female, æt. 38 years, had suffered from severe rheumatoid arthritis for over six years. Vasomotor disturbances began at fourteen years of age with frequent attacks of "dead fingers." The terminal phalanges were chiefly involved at first, but later the "deadness" would go up to the knuckles. At first the attacks generally came on after a long walk. She got to expect them when she became tired or fatigued. The condition in the fingers was always preceded by a "curious feeling of faintness"; there would be a kind of mist over her eyes, and then the fingers felt numb and assumed a whitish-yellow, or at times a mottled colour. The nail-beds assumed a sort of purplish hue. During each attack, which lasted generally about two hours, she was overcome by a "peculiar heavy feeling." The feet felt cold and numb, and it was often necessary for her to get off to bed and have hot flannels applied to the feet to restore the circulation. She always felt weakly on the day following a severe attack. After twelve months these attacks decreased in severity and frequency, but continued off and on till she was twenty-seven years of age.

E. M. K—, female, æt. 25 years. Onset of rheumatoid arthritis at twenty-one, with swelling and pain in the proximal inter-phalangeal joint of the index finger of the right hand. In the following three or four months the condition rapidly spread, and by the end of that time had affected practically all the joints of the body. For three months before the onset she suffered much from coldness and numbness of the hands, which would go "dead-white" as far as the wrists. There would be no feeling in them at the time. The blanching of the fingers was followed in about twenty minutes by the hands becoming hot and tingling. At this time the attacks came on every alternate day. The attacks gradually wore off in severity and frequency after the onset of the joint pain, and at the time of admission into hospital the only symptoms were coldness of the hands and feet, with frequently recurring attacks each day of clammy sweatings of the palms and soles.

M. L. W—, male, æt. 11 years. Onset of rheumatoid arthritis at age of 10 years in the right knee. Three weeks later the right wrist and knuckles became affected, then the elbows, ankles, and other joints. For a month or so before the hands became affected the fingers as far as the knuckles would go numb and white, and he would not know if they were moved about by another person. The fingers would go "dead." The whiteness and numbness was followed by congestion and tingling. He has never had these sensations since the joints have been affected.

M. R—, female, æt. 42 years, mother's help. Last year, for a few months before the onset, the fingers used to go "dead" after washing in cold water. There have been no such symptoms since the onset.

E. A—, female, æt. 52 years, dressmaker. Had blanching of the fingers for six months before the onset of the rheumatoid arthritis. These symptoms have lessened since the onset of the joint condition.

E. M. K—, female, æt. 25 years, factory worker. For three months before the onset the whole hands went numb, cold and bloodless as far as the wrists. Each attack lasted for about twenty minutes. The seizures came on three times a week on an average. These symptoms have disappeared since the onset of the joint condition.

K. P—, female, æt. 27 years, housemaid. There

have never been any syncopal attacks, but since the onset of the joint condition the fingers have been subject to congestive attacks, when they assume a purplish hue. The condition generally lasts several hours at a time.

E. B—, female, æt. 58 years, married. Five weeks before the onset in the knees she experienced tingling and congestion of the hands and feet. She had to cease washing clothes on account of the hands becoming so bloodless each time she got her hands wet. There have been none of those symptoms since the rheumatoid condition has become fully established.

S. B—, female, æt. 36 years, house duties. For two years before the onset she had marked Raynaud-like symptoms affecting all fingers up to the knuckles. Whenever it was the least cold "all the blood would go out of the fingers," and be followed by numbness and tingling. It was her custom to soak the hands in hot water containing soda, when the congestive stage would come on with a sensation of warmth and tingling. Since the onset three years ago these attacks have been very infrequent, and only affect one or two fingers.

That the relation between the initial vasomotor changes and the later joint swellings is a close one is illustrated in many of these cases. I have observed the joint changes localised to one or two fingers in each hand, which previously alone had exhibited vaso-motor disturbances. In some cases after the initial vaso-motor paroxysms the articular swellings may subside, only to reappear in the wake of an exacerbation of the vaso-motor symptoms. But the joints tend after a time to become permanently enlarged. So also the duration of each paroxysm varies within wide limits. In mild cases the slight syncopal changes are of only a few minutes' duration, and reappear only after an interval of several days. In the more advanced and severe cases each paroxysm may take three or four hours in which to complete the cycle of changes, and in the most severe cases the vaso-motor paroxysms may occur regularly two or three times each day in all seasons, frequently passing from one paroxysm to another without any appreciable interval. During such paroxysms the patient experiences the usual coldness and numbness. There is a blunting of the sensibilities in the areas affected. A lowering of surface temperature is observable during the syncopal period, while during the subsequent asphyxial stage the surface temperature again rises, this stage being followed by a clammy and often profuse sweating over the localised areas. During the asphyxial stage the nails present a slatey-blue colour. These "dead fingers" in rheumatoid arthritis are frequently symmetrical. Thus only one or two corresponding fingers of opposite sides may be involved.

In the later stages of rheumatoid arthritis there is observed an asphyxial condition over joints which are the sites of periarticular swellings. This condition is more or less present in all cases, and has been described by Bannatyne, Garrod and others. They ascribe the vaso-dilation to the pressure of the exudate in the periarticular tissues on the small venules bringing about backward pressure. The asphyxial stage of the paroxysms is not so clearly defined as a second stage as in true Raynaud's disease. In even extensive and complete pallor of the extremities there may be no succeeding duskiness of the skin. Moreover, in the great majority of cases of vaso-motor disturbances in rheumatoid arthritis, there is no succeeding painful stage such as is described in Raynaud's disease. In those old-standing cases where the circulation is feeble there is a more or less constant feeling of coldness of the extremities. In such cases the skin over these parts is nearly always of a slaty-blue colour, and such patients suffer from considerable discomfort from a constant state of moisture, especially over the palms and soles. But the processes do not go the length of shedding of the epithelium. Even in severe cases of asphyxial attacks occurring in the course of rheumatoid arthritis we do not find the distension of the veins of the hands, forearms or other parts proximate to the extremities affected such as has been described in Raynaud's disease.

These symptoms are sometimes preceded by cramp-like feelings and disorders of cutaneous sensibility over the areas involved. Tactile sensibility is blunted. For example, one lady told me that she would pick up two pins and think that she had only one; also that if she pricked her finger there would be no pain at the time, but it would take quite a minute before the pain came on. There is often a contraction of the skin-tissues binding down the underlying subcutaneous, muscular, tendinous, and joint tissues, till the fingers

become quite stiff, not from peri-articular fibrous ankylosis, bony or other organic changes, but because the enveloping skin-tissues are so tightly drawn that they will not permit of flexion. Pigmentation and localised œdema are other conditions resulting from the altered nutrition of the parts.

Desquamation occurs in severe cases, and is the result of the interference with the nutrition of the skin by the frequent and prolonged spasms of vaso-constriction. Degenerative processes take place in the skin-textures as the result of trophic and vascular disturbances. It becomes thinned, dried, glossy, hairless, and losing its elasticity. These changes may go the extent of bringing about death of the tissues with extensive shedding of the epithelium.

The transient obscurations of vision as described by Raynaud, Galezowski, Beaumont and Llewellyn in connection with this disorder may be explained by the vaso-motor changes in the retinal vessels, which symptoms are also mentioned in the notes of the first case quoted in this paper.

These vaso-motor changes of rheumatoid arthritis are the rule in women and children, but in men they are exceptional. In women who have no vaso-motor disturbances the earliest symptom is a feeling of stiffness in the fingers. In the most acute forms of rheumatoid arthritis, such as those in which the onset follows immediately after confinement, vaso-motor symptoms do not, as a rule, occur—the condition first manifesting itself by pain and swelling of the joints. Where the usual vaso-motor symptoms are wanting there are generally other nervous phenomena such as "pins and needles."

The general state of rheumatoid patients may be described as "ill-nourished." Their whole tone is lowered. The circulation is sluggish and they are very prone to chilblains and coldness of the extremities. The symptom-complex of these vaso-motor disturbances all tend to the view that there exists a toxæmia affecting the trophic nervous system.

July 28th, 1913.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from page* 240.)

(233)

The Veins of the Neck in Heart Disease.

The next thing to look for is the state of the veins in the neck. You know from the old statement that if you want to gauge the condition of the left ventricle you should feel the pulse, and if you want to gauge the condition of the right ventricle look at the veins of the neck. They are distended, you see.

("With Dr. Bradford in the Wards of University College Hospital," 'Clin. Journ.,' vol. xxxiii, p. 65.)

(234)

More Diagnostic Cloaks for Ignorance.

Whenever a patient complains of pain always be very careful before you attribute such a thing to hysteria. Most of these severe pains of which patients complain have got some real cause for them, and when you are not always smart enough at first to ascertain what the cause of the pain is, do not cover your lack of knowledge up by labelling the person as neurotic or hysterical.

("With Mr. Bland-Sutton at the Middlesex Hospital," 'Clin. Journ.,' vol. xxxiii, p. 97.)

(235)

Carbohydrates in Dyspepsia,
or
A Hint for Vegetarians.

As Professor Allbutt says—"There is no superstition more tenacious of life than that which prescribes carbohydrates to all dyspeptics as 'so digestible,' and into weak stomachs ready to dilate is thrown a mass of such a dish as rice pudding—a bulky food, imperfectly salivated and peculiarly apt to decompose with the disengagement of volumes of carbonic acid gas."

("Atonic Dilatation of the Stomach," by Ernest Young, M.D.Dunelm, 'Clin. Journ.,' vol. xxxii, p. 369.)

(236)

Vaccines an Addition to, but not a Substitute for, our older Therapeutical Measures.

First and foremost let me impress upon you the fact that vaccines have not superseded all other means of treatment: the general does not get rid of his infantry because of the accuracy of the fire of his artillery, nor has the torpedo superseded the big gun. There is just as much, nay, more need of accurate diagnosis as there ever was. It is the only true basis of treatment. What we all need to learn —and it is only experience that can teach us—is when we may rely on our old pop-guns, and when the new weapons are to be called in to supplement them. If you have ever seen a case of cystitis treated by vaccine alone, and then had the advantage of seeing the same patient subjected to the influence of bed, diet, diluents and urinary antiseptics as well, any doubt which you may have of the value of these latter will soon vanish. Don't ignore them. They may effect a cure whilst you are waiting for your vaccine.

("The Use of Vaccines," by W. Bruce Clarke, F.R.C.S., 'Clin. Journ.,' vol. xxxii, p. 321.)

(237)

Chronic Rheumatism,
A Diagnostic Refuge for a number of painful somatic Ailments.

Dr. George Oliver, speaking on the pathology and balneology of chronic rheumatism, says—"The so-called 'rheumatisms' met with in a mineral watering place constitute a somewhat heterogeneous group of painful somatic ailments open to some latitude of interpretation when viewed from the diagnostic standpoint."

("Discussion on Rheumatism," 'Clin. Journ.,' vol. xxxii, p. 310.)

(238)

Chronic Rheumatism and Fibrositis.

Over sixty years ago Sir Thomas Watson wrote: "It (rheumatism) is inflammation of a particular tissue—the *fibrous tissue;* and it may therefore manifest itself wherever that tissue is employed in the fabric of the body. . . . Rheumatism, therefore, is essentially inflammation of the fibrous tissues." Sir William Gowers, in his most instructive lecture on "Lumbago: its Lessons and Analogies," fixes this idea in the term "fibrositis," which he suggests as a designation for all the common forms of rheumatism and unilateral neuritis. I think this term is useful; it not only clearly indicates the site and nature of the trouble without committing us to any uncertain theory of causation, but it conveniently links together some apparently diverse pathological states into a comprehensive clinical entity, the co-existence of which is a matter of daily observation—such as *local neuritis, local fibrosis of arteries, gout, chronic rheumatism* and *senescence* along with *peri-articular* or *muscular* or *tendinous fibrositis.* All newly coined terms are apt to be resented as clumsy and uncouth; and that is the only objection which has hitherto been raised to the acceptance of the term "fibrositis."

(*Ibid.*)

(239)

Some of our many Limitations.

It is impossible for us to make a dwarf grow to a Sandow or an imbecile become a Darwin. They have not the requisite capital. Similarly we cannot make a testicle grow to full size and maturity if it is not so designed and capitalised.

("The Surgical Treatment of the Imperfectly Descended Testis," by Edred M. Corner, M.S., F.R.C.S., 'Clin. Journ.,' vol. xxxii, p. 303.)

(240)

Name-Plates,
or
Unreasonable Expectations as to the Specificness of Pathogenic Micro-organisms.

Dr. Poynton, in a discussion on rheumatism, said: "With regard to a specific micro-organism, so many bacteriologists speak of this specific micro-organism as if, could you only magnify it enough, you would be sure to see the word 'rheumatism' written on its back. These organisms are not so specific."

('Clin. Journ.,' vol. xxxii, p. 302.)

(241)

Hysteria, not Exclusive.

You may say, such a man as this (a butcher and farmer, æt. 39 years) is not at all likely to have hysteria, but you must remember that even the most robust are liable to it; it is not confined to one type of person, nor to one sex.

("With Dr. Herringham in the Wards of St. Bartholomew's Hospital," 'Clin. Journ.,' vol. xxx, p. 204.)

(242)

The Bacillus Coli as a cause of Puerperal Fever.

The colon bacillus, like the poor, is always with us, if not actually in the genital passage at least in very close proximity to it, and it is a well-known fact that damaged tissue in close proximity to the bowel very soon becomes infected by this organism.

("On Puerperal Fever," by Victor Bonney, M.S., M.D., B.Sc.Lond., F.R.C.S., M.R.C.P., 'Clin. Journ.,' vol. xxxii, p. 289.)

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DIAGNOSIS IN ABDOMINAL EMERGENCIES.*

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I ASK you to consider with me for a short time the diagnosis of abdominal emergencies. Diagnosis, and early diagnosis, is so difficult and so necessary. In no class of cases are the bad results of delay more apparent. Early treatment is so desirable, and early treatment presumes early diagnosis. Abdominal emergencies are common, and present themselves frequently to all of us. Elaborate methods of diagnosis are inadvisable, for they mean delay. Mistakes in diagnosis are frequent. It is to the diagnostic signs in early stages of the disease that I shall chiefly invite your attention. Treatment should not wait for the later signs, which may be the signs of impending death.

I am going to speak almost entirely from personal experience. To this, and to the extent of the subject, all omissions must be credited. The experience, however, has been considerable, for the cases bulk largely in hospitals and in general surgical practice. I will first speak as to how one learns the history of and investigates a case, and then I will deal with the diagnosis of the different emergencies separately.

How does one start out when called to an abdominal emergency? Promptly (even if told there is no hurry), with an open mind and with a pair of rubber gloves. A diagnosis over the telephone should not interfere with an open mind. A surgeon is not merely a carpenter: he must concur in, or take his share in arriving at, the diagnosis before he assists in the treatment. It is the height of affectation when a surgeon looks at a stethoscope as if he did not know one end of it from the other. I take my own stethoscope, because

* Read at the Newport Medical Society.

I can't use other people's; I take a skin-marking pencil, and this completes the equipment. It is not exactly a matter of diagnosis, but I also ask, if I am going any distance, if I shall bring the operating things and a nurse. This has saved several lives.

The history of the case.—After hearing this from the medical attendant, I get a direct history from the patient, if the patient is not too ill. The observations of friends are sometimes of value.

Onset.—I ascertain the time of onset, and was it sudden or gradual? The absolutely sudden, dramatic onset of some emergencies is quite characteristic. Is the onset attributed to anything? Food, cold, exposure, or over-exertion? What was the patient doing at the time of onset? Where did the pain first appear, and where has it subsequently shifted to? The nature and intensity of the pain? Whether the patient has been keeping still or sitting up or walking about? To what places, if any, does the pain radiate? Has there been premonitory "indigestion"? Is the emergency due, or attributed to, an injury?

Vomiting.—Has there been any? The time of its commencement? The frequency, amount, and character of the vomit? Whether much belching of wind? Whether much or little straining when vomiting? Whether any hiccough?

The bowels.—When did they act last? The amount, the consistency, and the colour of the motions, and any abnormalities observed in them? Often more important still, when was wind passed last, and whether it was the result of an enema or otherwise? Was it wind that smelt? Whether enemata have been given? If so, how many? When, by whom, what was used, and what results? Has there been any diarrhœa?

Micturition.—Frequency? Normal amount or scanty? The appearance of the urine? Any pain on micturition, and if so, at what part of the act?

The abdomen.—Has it altered in shape or in size? If any swelling present, when was it first noticed and has it increased?

Cough.—Present? Its character? Any expectoration? Any pain on deep breathing.

The record of the pulse and temperature.—If temperature is stated to be normal, whether it has been taken in the evening? Whether it has been taken in the mouth or in the axilla? Whether the patient has felt hot or been sweating? Whether there have been any feelings of chilliness or actual shivering attacks?

Any treatment?—Aperients? Enemata? Morphia? Other medicines? External applications? Results of treatment?

Any remission of symptoms?—This must be carefully inquired into.

Any diagnostic investigations which have been made?—The blood (Widal's reaction; leucocytosis)? The fæces? The urine?

If a woman.—Any children? Married or single? Number of pregnancies and date of last? Particulars of miscarriages? Facts relating to menstruation?

Previous illnesses likely to relate to the present.—Guided by what I have learnt, I make a few inquiries under this head. Whether any previous illnesses like the present one? Attacks of colic, of indigestion, of stomach trouble or of biliousness? Whether habitually constipated? Whether food bolted, or has suffered from bad teeth? Whether ever jaundiced? Whether in robust health up to the beginning of the present acute illness, or whether there has been some months' failing health and getting thinner?

The hygienic surroundings.—Particularly as regards drains, and whether other people have been ill in the house are sometimes worth inquiring into.

Alternatives.—Much of the above relating to the history of the case may be omitted where the diagnosis is clear, but all of it should be in the investigator's mind. Where the patient is very bad an immediate examination is best. With young children much of the history can be obtained in another room before seeing the patient.

The examination of the patient.—Get the hands thoroughly warm in hot water. The patient is brought to the edge of the bed and made as comfortable as possible and the examiner sits down. The facial and general appearance and the degree of strength and consciousness of the patient are noted and the pulse felt, so that an idea can be obtained as to what amount of investigation can be stood.

The abdomen is thoroughly exposed, the clothing and bed-clothes are neatly arranged, got flat (great lumps of blanket and eiderdown over the pubes obstruct examination) and isolated between clean towels. The classical order of inspection,

palpation, percussion and auscultation is then adhered to.

Inspection.—Distension or sinking in, visible coils or peristalsis, fulness in the flanks, colon and cæcum dilated or not, does the wall move well or is it held rigid, are there localised areas of relative immobility? Is the breathing shallow or rapid? Is there any localised prominence? Is there a visible viscus or tumour, and does such move up and down on breathing? Note discoloured, tumid or red areas and sites, and results to skin of poultices, blisters or other applications. Are the superficial veins dilated? Get the patient to point out the site or sites of pain, where it started, where it now is, and what is the very worst spot? (to be shown, if possible, with one finger).

Palpation.—Light at first. Start where you think it is not tender and reach the tender spot last. Map out extent of tenderness.

Note extent and depth of rigidity, and localised rigid areas.

Palpate beneath the ribs, in the flanks, on deep breathing.

Have the head and shoulders raised and supported, and see that the patient does not bob the head up or talk. Have the knees drawn up and the mouth opened. Use deep palpation and bimanual palpation. For the latter, sometimes it helps to hook one hand and arm right under the patient. It is also sometimes useful to lift the knees with the arm under them, tilting them towards the abdomen, so that the buttocks are partly raised from the bed while the other hand palpates the abdomen.

Note any areas of œdema of the abdominal wall.

Mark the lower border of the ribs, tumour areas, tender areas and suspected spots with skin pencil.

Examine the inguinal and femoral rings.

Remember distended bladder and pregnancy.

Percussion.—This should be light and deep, Abnormal areas of dulness should be mapped out and marked with pencil.

Auscultation is of little value. One can hear gas moving, and the significance of any bruit heard is to be considered.

Occasionally it is valuable to turn the patient on to the side or face.

Rectal and vaginal examinations.—A rectal examination should very seldom be omitted. Use a rubber glove, for a rubber finger cot does not sufficiently surely prevent infection of the hand.

Examine with the patient on the back (bimanually), and on the side. Search carefully for and note the consistence and position of any swellings. Find any tender spots. See whether the rectum is full or empty. See what the finger brings away, motion, blood, etc., and its character. Note any hæmorrhoids.

With regard to the vagina, any discharge and its character should be noted, and an examination made in married women and the genitalia investigated in the usual way.

Other parts of the body.—The chest should be examined, the lungs and heart. Inspect the mouth, the tongue and teeth. Note the smell of the breath. Examine the pupils. Ascertain the site of any headache.

Make a general inspection of the body. Are the feet cold or warm? Is there any œdema of the feet? What is the condition of the knee-jerks? Are there any indications of tubercle or syphilis, old or recent? Are there any glandular enlargements? Is there any rash?

Have the temperature taken. It is very useful sometimes to take it in the rectum, where it may be "up," although the mouth temperature is normal, or where it may be as much as two degrees higher than in the mouth.

Examine or find out about the urine.

I have spoken briefly as to how one approaches and investigates a case of abdominal emergency. I shall now take the various emergencies and draw attention to the leading symptoms in each, dwelling particularly upon the early symptoms and endeavouring to make what I say as practical as possible.

Appendicitis.—I shall start with the commonest emergency. It is always an "emergency." There is general abdominal pain, mostly epigastric, with probably an attack of vomiting. This is followed in a few hours by a cessation of the initial pain and marked tenderness, but often little or no pain in the appendix area. There may be nothing else, or there may be a slight rise of temperature, which is so transient as to be often missed. I believe that the first attack of appendicitis, which is probably rarely seen by a medical man, is always slight and is called "indigestion." In young children particularly it is nearly always passed

over. They cannot localise their tender spot, and it is supposed that their temperature goes up for slight causes such as "teething." In adults we have learnt to recognise the early attacks of appendicitis as appendicular gastralgia. But with the stomach symptoms appendix tenderness will often give the key to the diagnosis. The attack which brings the patient to the hospital or the consultant to the bedside is not, however, one of these first attacks; it may be the attack which undealt with, will lead to a fatal issue. How often has operation exposed an appendix, obviously the seat of old disease, when previous attacks are altogether denied?

Cases of ambulant appendicitis are not uncommon—even with abscess when it is well localised. Then there is back pain and a limp and the patient may be thought to have lumbago.

Contrast the two iliac regions on palpating.

During an attack remission of symptoms occurs. First there is acute general pain; then this subsides, and the pain settles into the right iliac fossa. Then the whole attack may subside or it may be only a remission. Then the next thing that happens is a progressive rise of the pulse-rate; this may be the only additional symptom, but usually there is a further rise of temperature, the initial tenderness becomes more marked, and there is great local rigidity and pain. These are danger-signals, meaning probably perforation or that the infection has spread from the appendix wall to surrounding structures. Subsequently the symptoms vary according to the degree and limitation of the infection. At first there may be a second short remission, the pulse-rate dropping and the patient feeling better; then either symptoms of abscess or of peritonitis. Let me remind you of the dictum that a case of appendicitis which has suppurated is a case which has been wrongly treated. If the dictum is right we must realise that either our diagnosis or our treatment is often to be condemned.

In the later cases, the foul breath, the tenderness, the lump, the swelling and tenderness on rectal examination, the back pain, the pain down the leg, the pain on micturition, the signs of peritonitis, the history of previous attacks are all diagnostic points. Vomiting is usual. The bowels usually do not act, neither fæces nor flatus passing.

You get diarrhœa in some early slight cases, and also in late cases with severe general infection, where it is a bad sign.

In young children, who as they lie in bed look very well, a very rapid pulse-rate indicates an acute general toxæmia. Such children not infrequently die in spite of operation, and an early recognition of cases of this type is particularly desirable.

Pus in the urine should not mislead. It may be present with appendix pus in the pelvic cavity. It is better sometimes to risk having mistaken a stone in the right ureter for appendicitis, rather than to delay treatment for an X-ray examination to be made.

Pneumococcic peritonitis. — This is difficult to diagnose from the peritonitis of appendicitis. It occurs most frequently in children, particularly in girls from five to ten years old. The symptoms are those of a rapidly spreading general peritonitis. Early diagnosis is essential, or at all events the arrival at an opinion that the abdomen requires opening. Lung symptoms may be present.

Acute cholecystitis.—Vomiting is persistent. The temperature is raised. There may be rigors. Pain is great. The local signs of peritonitis in the gall-bladder region with rigidity and tenderness are marked. There is back and right shoulder pain. Jaundice should not usually be expected; when present it may not be due to a blocking stone, but to an acute inflammatory swelling of the duct. So-called "biliary colic" is usually acute cholecystitis.

All perforations.—The onset is sudden. The patient falls as if shot. Such patients the moment previous to the perforation are apparently well; having tea, at work, perhaps in bed. There is much collapse and great pain. Then about twelve hours later the symptoms are much less marked This is the dangerous "latent period" and the condition may be missed, but the board-like rigidity of the abdominal wall, particularly over the perforation, remains. The patient in this condition may walk into a medical man's consulting room. There is often no rise in the pulse-rate at first.

Perforated gastric ulcer.—The patient is an anæmic young woman with a history of indigestion. There is absolutely sudden onset with marked shock. Then comes the latent period, but the great rigidity which is present from the beginning never goes. Absent liver-dulness is of no value; if found it is probably a late sign and should

not be waited for. Vomiting is more often absent than present, but the patient may vomit just after the onset. The temperature does not rise.

Perforated duodenal ulcer.—This is really an ulcer on or near the pylorus. With the sudden onset, pain and collapse, the rigidity and tenderness are most marked at the upper part of the right rectus; in a few hours this rigidity and tenderness may be most marked in the appendix area, the case simulating appendicitis. Vomiting occurs. Pulse and temperature are normal at first. The patient is a man between thirty and forty. There is rarely a history of duodenal ulcer symptoms; there may be a history of indigestion.

Perforated gall-bladder.—When the gall-bladder perforates from disease, the symptoms are if possible even more intensely acute than in the preceding classes, probably because of the highly toxic nature of the infected bile. The rigidity and tenderness are most marked over the gall-bladder. There is early, marked collapse, the pulse-rate goes up at once, and there is not likely to be a "latent period." With regard to history, seek for evidence of attacks of cholecystitis, and also find out if the patient has had typhoid fever, as the gall-bladder infection may be typhoid in character.

Perforated typhoid ulcer.—The sudden acute pain, tenderness and rigidity is usually in the right lower part of the abdomen. Early recognition is most essential. In typhoid hæmorrhage the rigidity is not marked and blanching and other signs of hæmorrhage occur.

Other perforations.—These occasionally occur. The subjects are usually past middle age. The site is the large intestine. Obstinate constipation, inflammation, thinning and perforation form the sequence of events. Perforation of cancers, stomach or large intestine occasionally take place. The symptoms are like other perforations, but the onset is more insidious and less dramatically acute.

Acute pancreatitis.—Acute general pain, suddenly coming on, with vomiting and no action of the bowels, so that the case resembles acute intestinal obstruction. There is epigastric distension and rigidity. The pulse is rapid. There may be irregular fever. Acute pancreatitis may be taken for anything.

Intestinal obstruction.—Early diagnosis depends upon—(1) constant, rather effortless vomiting; (2) abdominal distension and sometimes visible coils; (3) no passage of wind by the bowel. There is no rise of temperature, no rise of pulse-rate and often very little pain. The abdomen is quite soft, with no tenderness. The patient may be walking about. This is the time to catch the patient for operation. "Fæcal" vomiting is a late and very serious symptom. Enemata have no result, except that the first one or two may bring away scybala.

In obstruction of the colon from cancer there is a history of failing health and increasing constipation for some months; then acute symptoms suddenly supervene, vomiting and pain. In some of these cases a distended inflamed cæcum may give a picture like acute appendicitis: the temperature is up and the strictured colon may yet allow a little wind to pass.

Into the details of diagnosis between the different forms of intestinal obstruction I do not enter.

Remember always the hernial rings. Particularly Richter's hernia may be missed, or when found the patient may insist that it is not the cause of the symptoms and has been in the same condition for years, although operation later reveals it strangulated.

Mesenteric thrombosis and embolism.—There is a sudden onset with general acute pain, collapse and vomiting. The bowels usually do not act, and the symptoms resemble those of acute intestinal obstruction. There may be vomiting of blood or melæna.

Thrombosis may be associated with thrombosis elsewhere; embolism with heart disease.

An acute abdominal condition associated with purpura.—I introduce this because I have operated on two cases. The symptoms are those of acute intestinal obstruction. There is early collapse with a rapid pulse: there may be persistent hiccough. Vomiting is profuse. At operation a length of small intestine is found, swollen and red, the condition passing off in a patchy way above and below. Purpuric skin rashes and rheumatic pains are associated. There may be hæmorrhage from the bowel.

Pericolitis.—This may present itself as an acute condition, particularly in connection with the descending colon or the sigmoid. With the former it is like a perinephritic abscess, with the latter (diverticulitis) like an appendicitis on the wrong side.

Acute dilatation of the stomach.—The condition

may follow an operation. There is sudden onset with acute pain, resonant epigastric distension, vomiting, great collapse, rapid pulse and scanty urine. The stomach-tube withdraws large quantities of gas and fluid. The outline of the stomach may be recognised. The emptied organ soon refills.

Enterospasm.—This, when acute, may simulate more serious conditions. There is severe pain and tenderness, vomiting, collapse and constipation. Obstruction or peritonitis may be simulated. Distension is unusual, rigidity is not marked, constipation is not absolute. The patient is neurotic, and there is a history of many previous attacks.

Internal hæmorrhage.—It is worth just grouping in our minds those conditions where it is loss of blood causes the serious symptoms. Blood is vomited in cirrhosis and gastric ulcer. It is passed by the bowel in duodenal ulcer and in typhoid fever. Aneurysms may burst into the bowel or into the abdominal cavity. Injury, tearing solid viscera or ruptured extra-uterine fœtation produce intra-abdominal hæmorrhage. In all these there is pallor, rapid sighing, breathing, rapid, feeble pulse, subnormal temperature, cold extremities, perspiration, vomiting, great collapse and weakness and semi-consciousness. The abdomen is tender, moderately rigid, and may be distended. I pass on now to emergencies in connection with the female genital organs.

Acute salpingitis.—This, when it presents itself as an abdominal emergency, is usually perforative. The primary infection may, but can by no means always, be traced. The symptoms are like those of acute appendicitis, and on the right side the two diseases cannot be distinguished, but the differentiation is of no practical value.

Ruptured extra-uterine fœtation.—Here the symptoms of intra-abdominal hæmorrhage predominate. There is sudden onset with collapse, rapid, feeble pulse, pallor and breathlessness; pain, tenderness and rigidity are present in the lower abdomen. The temperature is subnormal. There may be a history of a missed period.

Torsion of the pedicle of an ovarian cyst.—This frequently occurs in pregnant women. The cyst is usually small, and often cannot be felt. It is particularly unlikely to be felt in advanced pregnancy. The condition is often diagnosed as appendicitis. There is an acute onset, but not so sudden as a perforation. Pain is out of proportion to the other symptoms; so great is it that large quantities of morphia are often given, and the patient begs for operative relief. Vomiting occurs, the bowels are confined, the temperature is normal or slightly raised, the pulse is good, with little, if any, increase in rapidity. There is lower abdominal tenderness, rigidity and swelling, much resembling a late appendicitis, but with these symptoms the tongue remains moist, and the breath is not foul.

Results of attempts to procure abortion by local means.—This must always be borne in mind as a possible cause of lower abdominal peritonitis in women.

Torsion of pedicles.—Besides the pedicle of an ovarian cyst, that of the spleen and those of other abdominal tumours may be twisted and give rise to acute symptoms.

Symptoms due to gonorrhœa.—An acute gonorrhœal peritonitis may occur in women. In men, inflammation of the cord may give rise to acute symptoms in the lower abdomen. This class illustrates the importance of a general examination of the body.

Abdominal injuries.—The parietes I assume to be uninjured. The fact of the injury, its mode of production, the time of its occurrence and the time, if any, which elapsed between the injury and the appearance of symptoms, must be ascertained. Generally the diagnosis has to revolve round the questions: Are the contents injured? If so, is there hæmorrhage from injury to a solid viscus or blood-vessels? Is there a tear of a hollow viscus with consequent commencing peritonitis? Or, are both these conditions—hæmorrhage and peritonitis—present? Where there is severe hæmorrhage, the characteristic symptoms—collapse, pallor, etc.—are immediate, with pain and tenderness most marked over the injured structure, liver, spleen or omentum. Slight improvement on being got to bed should not deceive. With torn viscera, the symptoms are more delayed and the patient may walk or even travel, and may not at once present himself to a medical man. Rigidity, however, comes on at once and is persistent. Like perforation of an ulcer, there is acute initial pain which may be followed by a "latent period." Definite symptoms of peritonitis should not be waited for.

Ruptured aneurysm.—An aneurysm may rupture intra-peritoneally. The symptoms are those of intra-abdominal hæmorrhage.

I consider now for a moment diseases not in the peritoneal cavity.

Acute pyelo-nephritis.—The patient is often a pregnant woman. There is a sudden onset with pain in the loin, running down into the iliac fossa, where it is very marked; tenderness, abdominal distension and pain, vomiting and raised temperature. There may be rigors. On the right side the condition may be mistaken for appendicitis. There may be only a little albumen in the urine and no micturition disturbances. *B. coli* is found in the urine.

Renal colic.—Rigidity of the lumbar and iliac muscles may suggest an intra-peritoneal catastrophe, but the intensity of the pain with its situation is usually characteristic. Pain is in the loin, radiating to the groin, testicle and thigh. Perhaps hæmaturia is present. There is vomiting and a very coated tongue.

Dietl's crises.—These are attacks of acute pain associated with movable kidney and supposed to be due to kinking of the ureter. There is vomiting, prostration and general abdominal pain. There is often hæmaturia. The movable kidney may be felt and is tender. The history of previous attacks is of value in diagnosis.

Acute symptoms in association with enlarged prostate.—Pain and tenderness in the lower abdomen, perhaps unilateral, vomiting, pyrexia and a coated tongue may be due to an attack of acute congestion in an enlarged prostate. The retention of urine may be only partial, and with such partial retention, the distended bladder may form a tumour, perhaps unilateral. Such cases may mislead, and call for careful investigation.

Osteomyelitis of the spine and acute arthritis of the hip may simulate acute abdominal conditions.

Other diseases which may mislead and which are not surgical I can only enumerate. These are: Acute gastro-enteritis, pneumonia, diaphragmatic pleurisy, typhoid fever, crises in tabes dorsalis, tuberculous meningitis, infective endocarditis, lead colic, poisoning, particularly from copaiba, turpentine and abortifacients.

In conclusion I would only emphasise what I said at the beginning. Let the diagnosis be an early diagnosis, *i. e.* arrived at from a recognition of the early symptoms so that it may lead on to early treatment. A mistake in diagnosis may sometimes result in the unnecessary opening of a healthy abdomen. This, with modern precautions, should have no bad result. Such a mistake is as nothing compared with the fact of the thousands of lives which our early treatment will save.

August 4th, 1913.

A DEMONSTRATION OF SURGICAL CASES.*

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Sarcoma of the Femur in a Child.

This first patient is a boy, æt. 12 years, who came up to the hospital with the history that this swelling of the lower end of the femur had been present for one month. When you examine it you find that the swelling is entirely above the knee-joint. The patella is quite movable, the leg is curved perhaps a little backwards, and on the femur, just above the knee-joint, is a large hot swelling, and on first touch it seems as if there was œdema over it. But although the covering feels like œdema it does not pit like œdema, neither does it fluctuate. The swelling goes all round the femur; notice, it is not only on one side, but in front, on both sides and behind, and that it imparts to your hand a tense elastic sensation. That swelling, of course, might be one of two or three things. Its rapidity is against the probability of its being connected with caries of the femur. But its hotness and its spread are suggestive of it being due to some septic osteomyelitis, that is to say, acute bone disease. The suspicion that it may be due to septic or infective bone disease is strengthened by the situation of this swelling. We know quite well that suppurative periostitis causes swelling, particularly in this situation at the lower end of the femur, and as a rule we open those swellings through the inner side of the popliteal space. Such cases of acute bone disease, as they are called, are associated with a short history, a red swelling and a painful swelling. You notice the swelling in this boy is not painful, allowing us to handle it freely, as you can see. And in the case of acute bone disease it would be associated with a temperature, and this boy has no temperature. So that the diagnosis of acute infective bone disease does not entirely suit the case, just as we have seen tubercle did not fit it. And when we look round for X rays to help us you see that in this skiagram, for instance, there is very little, except that just in one place the femur seems at a slight angle, the periosteum is a little raised, and

* At the Polyclinic.

there is a little new bone formation. The next skiagram is rather better, because in it you see on both sides of the femur the periosteum has been raised, and there is a little new formation of bone *in* the swelling. That is very important. It is very unusual in young children to be able to see new-formed bone in the skiagram from a periosteum which has been stripped up by pus, and the fact that we can see that new-formed bone in this is suggestive that the swelling is not pus. It suggests that in this instance we are dealing with sarcoma of the lower end of the femur. The growth in this position is of only one month's history and is hot. Therefore, we shall have to argue that it is a rapidly growing sarcoma, accounting for the shortness of the history and the heat which has developed in the part. This diagnosis of rapidly growing sarcoma is also supported by the fact that there is a tense elastic swelling of the thigh here, with some dilated veins over the inner side, which you can see well, and owing to its painlessness you can trace the swelling right up to the normal femur, and in that way you can feel where the swelling ceases and the normal bone begins. The fact that you can trace the swelling from the abnormal part to the normal femur is very greatly against acute infective bone disease, because in either acute or subacute infective bone disease the part is too tender to allow you to trace the swelling from the abnormal to the normal. Further than that, in infective bone disease you do not have so sharp a limitation between the normal and the abnormal as in this case. So I think in this case, humanly and reasonably speaking, the diagnosis of rapidly growing sarcoma of the lower end of the femur is as sure as it can be in children. But in children the diagnosis of rapidly growing sarcoma is always a matter of considerable difficulty. In the first place, in children, these tumours are round-celled, and it is very unusual for the periosteum to be stripped up to any marked degree, and it is very unusual for bone to appear in the middle of a sarcoma in a child like it does in the case of an adult. As is shown in the skiagraphs before you of sarcomata in adults, a plenteous supply of new-formed bone growing in the midst of the sarcoma is shown. In the rapidly growing sarcomata of children you do not find that. In consequence, the diagnosis in children is always complicated by two questions: (1) If the sarcoma is at all of a slow-growing variety, the diagnosis of tuberculous disease is much more likely to be true than sarcoma. (2) If, on the other hand, you are dealing with a rapidly forming swelling, then the consideration of subacute infective disease of bone complicates the diagnosis. And further, one would point out that in dealing with these swellings in children you have comparatively little help from the X rays, as shown in this picture. As a general rule, the skiagraphs are bad; the bones are not well shown and the margins are not well defined. In the case of sarcoma the margins of the bones are not well defined because of the growth, and in the case of infective bone disease the margins are not well defined because of the pus which is present. So that, in dealing with these swellings in children, the diagnosis is not much aided by the X-ray picture. And therefore, in all these cases, considering the element of doubt that exists in connection with them, and remembering one's own fallibility, it is much better to make an exploratory incision before you begin the radical treatment. And that is what will be done in this case. In the case of infective bone disease we get pus, and in the case of sarcoma you will have hæmorrhage on making the incision. And though the result of the exploratory incision is not absolutely certain—for it is conceivable that there might be bleeding only from granulations in a septic case, and an appearance of pus from a breaking-down growth—you have to decide on that, because you have little or no help from the X rays. And if you take out what you regard as a piece of the tumour and have it submitted to a skilled microscopist for a report, again you are not in the least certain of the result. I have seen, more than once, a pathologist of position diagnose a granuloma as a sarcoma; and one may say the more skilled the pathologist is, the less is he likely to give a certain diagnosis. So you may take it that, in dealing with these sarcomata of children, the pathologist, like the X-ray man, is more likely to fail you than he is to be of any particular use, and that therefore you must rely almost entirely upon yourself, as a result of your clinical examination and the exploratory incision which it is necessary for you to make, to decide how you shall act. And I would point out, in regard to this boy, that in adults, particularly in the case of rapidly growing

sarcomata, there is a great liability to spontaneous fracture, whereas in children that is not so. Sarcomata in children, although they may originate from bone, grow rapidly *outside* the bone, and the bone itself is not weakened, and so you do not have spontaneous fracture.

Granulomata of the Jaws.

This next patient, a little girl, I bring after the previous patient because she presents clinically a very similar question. Some months ago—I do not remember exactly how long—she came up with a very curious condition of mouth, in which there was hypertrophy of the gums, the gums growing up above the teeth. We went into all the different questions, such as improper feeding, scurvy, pyorrhœa, and so forth, which would produce swollen gums, but it was not any of them. Some of these granulomata, as they were supposed to be, were taken away and examined by expert pathologists. Number 1 pathologist said they were sarcomata. Pathologist Number 2 was called in to confirm that, and I think he did confirm it. At any rate, Number 3 pathologist, who was probably the most skilful of all, showed his skill by hedging; and he said that it seemed like a sarcoma, but the microscopical appearances could bear a different interpretation. That means that they might be inflammatory lesions. That was a very important question, because this little girl at that time was very unlike a case of sarcoma. She had this granulomatous growth of both upper jaw and lower jaw spreading over the teeth. So, if it was sarcoma, it was sarcoma of both sides of both jaws, and hence there was nothing in the way of operative treatment to be done. So the dental surgeon acted on the supposition that they were infective granulomata, and they were all cleared away, and they have been cleared now for some months. Now, in the upper jaw, there is a return of the condition, which you can see just under the alveolus. I do not think there is any doubt that in this patient one was dealing with a non-sarcomatous condition, such as even skilled pathologists cannot be trusted to diagnose.

Tuberculous Mediastinal Abscess.

This next patient—a boy—has, I think, been here before. He is now æt. $4\frac{1}{5}$ years, and about three years ago he came to the Hospital for Sick Children, showing the shadow of an abscess in his posterior mediastinum. It was a difficult case, because the shadow of that abscess coincided more or less with the shadow caused by the heart. It corresponded with what might be called the shadow of the cardiac dulness. A costo-transversectomy was done on the right side. In that operation one takes away the transverse process of the vertebra and the hinder part of the rib, and so gets into the posterior mediastinum without opening the pleura. By that means an abscess in the posterior mediastinum was opened. The X-ray picture had shown that the shadow of the abscess was mingled with that produced by the heart. At the operation the anterior wall of the abscess was found to be the posterior wall of the heart. In consequence, one could not curette it, or do anything except sew it up. The wound remained healed for some time, and then broke down. Later, there was a secondary infection, and the sinus went on discharging for some considerable time—something like two years, I think. Lower down he had another abscess, which did very well indeed. It has healed up entirely now, and bears a tale of the method of healing up. You know, people ascertain the shape, size, and direction of a sinus by injecting it with bismuth paste and taking a skiagram, which, of course, shows the bismuth paste. It was the X-ray people who used to do this, and in that way they discovered that a certain number of sinuses began to heal up after they had injected them with bismuth paste. This patient shows it is a curative method, but it is not quite so good a method as what I have just said to you might lead you to imagine. In the lower part of this boy's back you can demonstrate a small abscess, and that small abscess you can see in this skiagraphic plate. So the tendency in these cases is for the main sinus, which has had the injection of bismuth paste, to heal, and for the injection to act as a cork—not letting the pus out of the main sinus, so that it has to find its exit somewhere else; and so we find this abscess down here. That does not cause him the least discomfort or trouble; he eats and sleeps well, and is now much better than he was before. The original abscess was caries of the spine. (The abscess mentioned was never opened, disappearing spontaneously. The boy was later shown, quite well, at the Children's Section of the Royal Society of Medicine.

Spastic Diplegia—"Little's Disease."

Here is a girl, æt. 10 years, who presents a comparatively common condition for us to come across in hospitals. And there is always one great difficulty in the treatment of these cases, so that from the point of view of treatment I would impress upon you one fact. A number of these children have very imperfect mental development, and so, from the practical point of view, I recommend you to divide them into two classes: to see whether you are dealing with a child who has good mental development, or with one who has only a poor mental equipment,; because it is worth while taking greater care and trouble in the treatment of the child with good mental development than with one of imperfect mental development. Children with backward mentality cannot utilise the benefit conferred upon them by the treatment. One has seen cases in which elaborate operations for lengthening tendons, and so on, occupying a long time, have been done, and at the end of that time one finds that the child has not sufficient mental development to learn to walk, and, in consequence, all the energy and effort which have been expended in surgery are wasted. This child is 10 years of age, but we are dealing with a case in which there is good mental development, and therefore one in which it is worth while to go to the labour of an expensive treatment. The pathology of that condition is this, as illustrated in this girl's history. As soon after birth as the people observe it, there is found to be paralysis and rigidity of the lower limbs, so that one can lift up either limb and find it to be rigid. Further than that, it is in a position of adduction and is kept rigid. That condition is followed by various contractures of the ankle, and, later on, of the hip, knee, and foot. The condition is what is called spastic paralysis, and appears directly after birth, and in two limbs; and, unless something is done, it will gradually become worse and worse. What was thought to be the true pathology was that there was a hæmorrhage in the cranium which was large enough to involve both leg areas. That pathology was largely an arm-chair pathology, because when people came to examine the brains of these cases they could not find these hæmorrhages. So that the view that the cause was hæmorrhage has exploded. Still, it was found that there was injury. Those who examined these brains found there had been thickening of meninges over the leg areas, but that did not account for all the cases. What they found far more universally was an imperfect development of the cortex cerebri. Why there was that imperfect development, so far as we know, there has been no light thrown on that. Certain—and one believes rare—cases are due to a hæmorrhage, but the more frequent cases are due to injury; and still more frequently you have this cortical lesion which results in imperfect development of the cortex, from what cause I do not know. But the result of that cortical lesion is very obvious. The arms and sphincters are all under full control, but there is this rigidity of legs with adduction, and spasm of these muscles of the calf, with contractures of the foot; and here there is what one does not always see, a tendency to chilblains. You would not expect that blueness of the toes and tendency to chilblains in a case in which the lesion was cortical. So this case presented by our little patient is good to illustrate that these cases of spastic diplegia, as they are called, have not all a uniform and simple pathology, but that possibly secondary to this lesion there are degenerative changes in the cord itself, which degenerative changes, in their turn, lead to certain trophic changes. We need not dwell further on the pathology, because it is obscure, and I cannot tell you anything more definite. But on the clinical side the case is perfectly well defined, and in this particular patient it is worth while to consider the treatment which can be adopted. I have utilised the opportunity of bringing the child here in order to think out the treatment to be pursued. The treatment which I am going to suggest is this: that one does a laminectomy, dividing certain posterior nerve-roots between the posterior ganglion on the nerve-root and the spinal cord. That is called, after the German who first did the operation, Förster's operation. The rule in regard to the division of the posterior nerve-roots is very simple: you are not to divide more than two consecutive nerve-roots. Having divided two, you miss one out; so, if you divide 1 and 2, you miss out 3, and you can then divide 4 and 5, but you must miss out 6. There have been surprising results from that. We have had well drilled into us, as students of physiology, that the posterior roots are sensory, so that by

division of these roots there should be a loss of sensation. But that is not so. That is a strange thing which we have learnt from this operation and its results ; at all events, there is but very little loss of sensation. But what you do find is that these patients have a loss of rigidity. This rigidity, which you can feel in this child, is not due to an irritation of the cortex ; it is due to some reflex influence, and by resecting the posterior roots you divide the reflex arc, which is active, and by that means you can get rid of all the spasm, or, at all events, a great deal of it. That sounds fairly simple, and it is quite easy up to the stage of having completed the laminectomy. What is difficult is the selection of the posterior roots which you shall divide. Because, as a matter of actual, practical fact, you have the greatest difficulty in deciding which is the first lumbar and which is the twelfth dorsal root. But even in that respect you can be happy, because a number of cases have now been done, and we know that it does not seem to matter very much which ones you have divided. That is the weakest part of this proceeding. You need to divide the roots, and it does not matter much, apparently, which you divide. The results of such operations were disappointing, in that the patient unusually realised, and rarely exceeded, our hopes of improvement. But a great deal could be done by the operation to make the sufferer from spastic diplegia capable of looking after himself, operation being the preliminary to education.

Deformities after Arthrectomy.

Here is a boy, æt. 4½ years. He had a tuberculous knee, for which arthrectomy was done ; you can see the incision, starting here and going round the knee. He did fairly well after that. But, as is too apt to happen in these cases, after a time an abscess appeared on one side of the scar. That was opened, and he developed a sinus, and after the sinus there came a secondary infection of the joint and further trouble. This occurred eighteen months after the first operation. These cases are very important, and very unsatisfactory to treat. It was thought that they would be very suitable for vaccine treatment, and we made vaccines from the cultures of these sinuses and had it injected ; but those vaccines, in my hands, were very disappointing. On Sunday I was at Treloar's Home, at Alton, and heard there that they found vaccines very unsatisfactory. When that line of treatment fails, you can try what I showed you in an earlier case—the injection of the sinus with bismuth. That sometimes fails ; it fails oftener than it succeeds. You will remember we saw in the other case that the patient developed another abscess, and there was some bismuth paste there. So here one opened the incision for the arthrectomy again, and packed the knee with a strip of gauze lightly coated with sulphur paste. The exact composition of that paste I do not, for the moment, remember. The sulphur in the paste meets with the fluids of the blood in the body, and forms a thin layer of sulphuric acid ; and that sulphuric acid will destroy any infection which is contiguous to it. That was done in this case, then, and in due time, when the cause was removed, and a little later, when all the sloughs caused by the sulphur had separated, the wound became full of excellent, healthy granulations, and it completely healed. Although the knee is healed up, we have, as the skiagram shows you, a fibrous union between the femur and the tibia, with the result that there is some little movement here at the knee-joint, and that is the difficulty with this boy. If there had been no movement, he would have had a perfectly strong and stiff limb. So the question arises as to what we shall do. I think the best thing is this. Put that knee up in plaster-of-Paris, which will extend from above the ankle-joint to a little below the trochanter, and hope as the boy grows up the knee will become more and more fixed. It will become fixed in this way. As he grows up, the healthy tissues in his body will grow quicker than will the pathological tissues ; namely, the femur and tibia will grow quicker than the fibrous union between them. So the fibrous union will appear gradually shorter and shorter, and, as the fibrous union becomes shorter, the leg will become more fixed. A particular form of splint is used at Alton, and is made of celluloid. People are afraid of that substance because it is inflammable. But although this material from which the splints are made is called celluloid, it is not the ordinary commercial inflammable celluloid. This material is not absolutely non-inflammable, but it is not so inflammable as ordinary celluloid. But it forms a strong, light splinting, which seems to be ideal in these cases.

Two Cases of Hernia, one with Movable Testicle.

These two little boys are both cases of hernia. But, although they are common inguinal herniæ, I

thought there were one or two things about them which one could profitably demonstrate. For instance, one of the most common difficulties in these children, when we see them, is due to the fact that they cannot cough in order to bring the hernia down and make it obvious. You may make the child run about, but the hernia may not come down after it, so we commonly have to diagnose on which side the hernia is without having seen it. There are a number of things to remember. I selected this boy because he shows very well the method I want to tell you about. A good plan to find out on which side the hernia is, is to get the spermatic cord under the finger, and just roll it from side to side. That does not hurt the child, and in that way you can feel on which side the cord is the thicker, and that is the side on which the hernia is. It is a very useful sign to know, and this boy shows it very well indeed. It is quite common for a child to be brought to one, and the mother and the nurse disagree as to which side the hernia is on. The case may be on the operating table and under the anæsthetic, and you find that the house-surgeon and the sister diagree as to which side the hernia is on. But, in nine cases out of ten, this method, namely, by feeling the spermatic cord, is a sufficient indication of the side the hernia is on.

The next boy illustrates the condition which is very confused with regard to these herniæ in children—the position of the testicles. The testicles may be high up, or they may be at the bottom of the scrotum. I may get a case admitted from the out-patient department labelled "right undescended testicle." When it comes to the ward the house-surgeon says it is on the left side. When you examine it yourself, you may find both testes are high up. That is common in children, particularly when associated with congenital abnormality. When I examined this child this morning both testes were high up, and now you find both of them are down in the scrotum. That condition of movable testicle is one on which one may be called upon to give an opinion. Far most frequently it settles down, as the patient grows up, with the testicle in the scrotum; but it is a condition which requires to be looked at, because it is usually associated with some abnormality of testicle and spermatic cord, and that abnormality is most often a congenital hernial sac. In consequence, whenever one sees this condition of testicle, and there is a suspicion of hernia, it is better to operate on that hernia. If you do that, it is not necessary to suture the testicle in the scrotum or do anything like that; merely replace the testicle in the scrotum.

August 4th, 1913.

CLINICAL NOTES ON SPONTANEOUS HÆMORRHAGES.*

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GENTLEMEN,—I propose to call your attention to-day to the subject of spontaneous hæmorrhage. It is important to recognise this variety of bleeding, and to trace out its ætiology and significance in any given case.

Spontaneous hæmorrhage is that which occurs without any obvious cause, such as an injury or wound. The significance of it varies greatly. The most marked instance of it is met with in persons who are the subject of hæmophilia or the hæmorrhagic diathesis. In such cases bleeding may occur from almost any point in the body, often without provocation, and readily after slight injuries or abrasions. These bleedings may be very copious, as in severe hæmaturia, or productive of arthritis when there is a large effusion into a joint. A remarkable feature is the tendency to a rapid reproduction of the blood which has been lost, and the further tendency to a subsequent growing up of the vascular plethora, which appears to relieve itself sometimes in renewed hæmorrhage from various points. So we may meet with epistaxis, bleeding from the gums, hæmatemesis, or melæna. The blood is defective in coagulative quality. In cases of grave, so-called "pernicious," anæmia spontaneous hæmorrhages may occur. The older physicians used to describe the condition of the blood in these and other kindred states as the "dissolved state" of it, there being a tendency to oozing and bleeding from various surfaces.

We meet with this condition as a terminal symptom in cases of cirrhosis of the liver, in which bleeding is apt to occur from many surfaces, independently of the hæmatemesis and melæna, which depend on portal venous obstruction. In the alcoholic variety of cirrhosis, the stigmata or angiectases, which are significant of it, are apt to bleed spontaneously or readily on slight provocation.

* A lecture delivered at the London School of Clinical Medicine, Greenwich.

In chronic jaundice, however induced, there is a hæmorrhagic tendency, and this is also met with in cases of acute atrophy of the liver (*icterus gravis.*)

Some of the most noteworthy spontaneous hæmorrhages occur in cases of chronic interstitial nephritis. The association of high arterial blood-pressure explains many of these. There is, of course, the liability here to cerebral hæmorrhage, more or less grave in any instance, but there may be epistaxis, hæmatemesis, hæmoptysis, and melæna, with or without intestinal ulceration. Arterial sclerosis, widely spread, may involve the kidneys, and if associated with gouty symptoms, may lead to varied sources of spontaneous bleeding. Metrorrhagia may be met with in some of these cases.

The skin-eruptions in connection with granular kidneys sometimes present a hæmorrhagic tendency. In minor degrees in such cases it is not uncommon to note oozing of blood from the gums and pharynx.

It is fairly certain that a copious epistaxis in elderly people with thickened arteries is a safeguard against a fatal intra-cranial hæmorrhage, and is so far salutary and protective for them.

Hæmaturia, sometimes recurrent, is a distinct symptom of granular kidneys. This occurs without any clotting, and, as Dr. West has described, it is probably often supposed to be of vesical origin.* This is also met with in attacks of oxaluria, when showers of octahedral crystals are formed in the kidneys, and shed along the ureters with considerable pain in the urinary passages.

Even in young patients epistaxis may be frequent, and depend upon early interstitial nephritis, with thickened arteries, cardiac hypertrophy and slight albuminuria. Occasional epistaxis in schoolboys should always be carefully noted and traced to its associated connections. The condition of the arteries and urine should be inquired into and the family history and the pulse-tension should be studied in such cases. Headaches are apt to occur in some of these patients. Instances, too, of orthostatic albuminuria in young lads and girls should also be regarded and watched with a view to such possible complications.

Mr. Armstrong, of Wellington College, believes that this connection exists not infrequently in the "cyclical" type of orthostatic albuminuria. In these cases he has noted that the systolic pressure falls on assuming the upright position, instead of rising as it usually does. The prognosis is not grave in such instances, but it is right to observe and follow up the clinical features of them for some years subsequently, if possible. In the "lordotic" or mechanical class of cases Mr. Armstrong finds no variations of blood-pressure, and the albuminuria is practically constant and not cyclical.

Hæmorrhagic purpura (Werholf) is attended by hæmorrhages, internal and external, which may result in epistaxis, buccal, gastric, intestinal, pulmonary, genito-urinary, retinal and cerebral bleedings, in addition to the characteristic cutaneous vibices.

Henoch's purpura, with severe abdominal symptoms, is also attended by various hæmorrhages, which may occur from the nose, lungs, kidneys, and into the adrenal bodies. An extravasation in the latter is a serious matter because it is apt to prove fatal from severe nervous shock, and, if not detected at an autopsy, might lead to a wrong interpretation of the immediate cause of death.

We may encounter spontaneous hæmorrhages in cases of leukæmia and splenic anæmia, also in advanced stages of scorbutus.

Retinal hæmorrhage demands a thorough examination of the circulatory system generally and of the condition of the kidneys.

The spontaneity of any sudden bleeding, where there is no obvious explanation of it, will often be found to depend on some more general, and sometimes remedial, constitutional fault which is revealed by careful study. There may be weak points here or there in the arterial tunics laid down in early life which are unequal to extraordinary strain in after-life, purely local. Elderly gouty subjects may suffer occasionally from attacks of slight hæmaturia, which subside after existing fears of calculous nephritis and cystitis.

Now, as to the treatment which may be necessary for some of these cases in which spontaneous hæmorrhage occurs.

Here, as always, prevention, if possible, is best. The management of every case of hæmophilia is persistently arranged to avoid even trifling injuries or strain. The life of such a subject is, more or less, an invalid one. Often, without any obvious

* "Granular Kidney, etc.," 'Lettsomian Lectures,' 1900, p. 103.

cause, hæmorrhages from any surface or into joints may occur. Adrenalin may be applied to bleeding surfaces. Compression should be applied to any external wound. Calcium lactate is useful in doses of 15 gr. or more, repeated as required. The nares may require plugging. Occasional doses of saline aperients are effective in preventing the general tendency to vascular plethora which is observed in some cases, and reducing blood-pressure.

In the majority of spontaneous bleedings, little active or immediate treatment is called for. In elderly subjects, with defective vessels, a regulated unstimulating dietary is important, with reduction of strong animal food. In purpuric cases, turpentine is probably the best remedy to restrain the hæmorrhagic tendency, and, failing this, adrenalin and calcium lactate may be employed. Nothing is likely to restrain the profuse bleedings (hæmatemesis and melæna) dependent on portal venous obstruction in hepatic cirrhosis. They are apt to recur and prove fatal.

Small hæmorrhages in elderly gouty subjects usually subside, and are best averted by appropriate diet, diluent liquids, and occasional gentle aperients to diminish arterial tension, if this is excessive.

August 4th, 1913.

Rupture of the Spleen.—Norrlin has compiled 13 unpublished Swedish cases of rupture of the spleen from a contusion. Two of the patients were women; the youngest patient was 7, the oldest 67, and the spleen was already pathologic in 4 cases. In one case the contusion occurred in an epileptic seizure, the spleen being already degenerated from alcoholism and infection. The first stage of shock and internal hæmorrhage is generally past when the patient is seen. The key to the diagnosis is an exploratory laparotomy, and it is better not to waste any time on exact differentiation. The results of operative treatment are growing constantly better; except for the first case (1899), the 9 patients operated on recovered after tamponning or splenectomy. In one case the rupture was sutured and tamponned, but persisting hæmorrhage made splenectomy necessary the next day. In two cases only part of the spleen was removed. Four of the five patients treated by splenectomy with immediate suture of the abdominal wall had regained full earning capacity when re-examined from two to nine months after the accident. Two boys, 10 and 11, both recovered after tamponning the rupture.—*Journ. A.M.A.*, vol. lxi, p. 150.

IONIC MEDICATION.*

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Ionic medication has been chosen as the subject of this lecture, for the reasons that its application does not require expensive electrical apparatus and that excellent results are often obtained by its use. In ionic medication we use an electrical current for the purpose of introducing drugs into the part which we wish to treat. In order that this method may be successfully applied, it is essential that the principles of the subject should be understood, otherwise failure, or even injurious results, will follow.

The chemicals that can be introduced into the body by the ionic method include the acids, alkalies, organic and inorganic salts, and salts of the alkaloids, providing that each one is soluble in water. When these bodies are dissolved in water, many of the molecules dissociate or divide into two parts, and each part takes a charge of electricity. These electrically charged bodies are known as "ions." The ions which take the positive charge are the ions of hydrogen, of the metals, and of the basic portion of the alkaloidal salts. The ions that take the negative charge are those constituting the acidic portion of the molecule. Thus sodium chloride dissociates into two ions, one, the sodium ion, bearing the positive charge, the other, the chlorine ion, bearing the negative charge. Sodium salicylate dissociates into the positively charged sodium ion and the negatively charged "salicyl" ion. Cocaine hydrochloride dissociates into the positively charged cocaine ion and the negatively charged chlorine ion.

If, now, an electrical current traverses the solution, the ions, which before were moving about in any direction, now take a definite course. The ions bearing the + charge are repelled from the positive pole (the anode)—the pole by which the current *enters* the solution. The ions bearing the negative charge are repelled from the negative pole (the kathode)—the pole by which the current *leaves* the solution. Thus the ions with the + charge travel with the current, "down stream," those with the — charge travel against the current, "up stream."

Besides the ions which are being repelled from

* Lecture delivered at the Polyclinic.

the pole there are the others bearing the charge of the opposite sign, and these are attracted to that pole. These last-mentioned ions, on reaching the pole, may give up their electric charge and appear in the free state, and then, perhaps, undergo further chemical change. In this way chemical products accumulate at the poles.

Suppose, now, that an electric current traverses the body by means of two wires which make contact with the skin. The tissue fluids contain ions, and those with the positive charge will travel with the current; many will reach the negative pole, lose their electric charge and form chemical products at this pole. These products may destroy tissue in the region of the pole. The ions bearing the negative charge will reach the positive pole and produce changes there. A practical application of this is seen in the destruction of moles, warts, hair-follicles, etc. The process is spoken of as "surgical ionisation."

But for the purposes of medical ionisation we interpose, between the skin and the wires conveying the current, pads soaked in the solutions containing the ions which we wish to introduce. Therefore if we wish to introduce zinc, lithium, magnesium, or cocaine ions, the pads containing them must be connected with the positive pole of the battery, because their ions bear a + charge and will thus be "repelled" into the body from the pad. So also, if we wish to introduce chlorine ions, iodine ions, salicyl ions, the pads containing their solutions must be connected with the negative pole of the battery. The chemicals which accumulate around the poles will dissolve in the pads and will not injure the tissues if their solutions are not allowed to become too concentrated. Hence the importance of using thick pads. We have now to inquire the fate of the ions that have entered the body. It must be remembered that the deeper they penetrate the further they spread. Some will enter blood-vessels and lymph-channels and be carried far afield; others may meet ions bearing an opposite charge, the two may combine and form an insoluble compound which cannot dissociate again and form fresh ions. So only the superficial structures can be effectively treated by ionisation, and if the deep-lying parts derive benefit, this benefit is due to the redistribution of their contained ions and not to the introduction of fresh or foreign ions from without.

The Practical Application of the Method.

For this purpose we require (1) solutions of the drugs, (2) pads to hold them, (3) cords to connect the pads with (4) a source of current.

(1) The solutions required should be dilute, not more than 1 or 2 per cent.

(2) The pads can be made of lint, not less than sixteen layers in thickness, or if a large area requires treatment, of folded towels of similar thickness. They must be well soaked in the solution and slightly wrung out, not too dry, but to prevent any solution from dripping from them when they are in position. If they are applied to the normal skin the latter had better be first washed with ether soap, in order to remove any grease (which would prevent the entry of the ions). The pad is then applied evenly, so as to make good contact with the skin. On top of the pad is placed some contrivance to which the cords may be fixed. A useful device is a piece of felt to which is stitched a piece of fine metal gauze. The end of the cord may be inserted under the gauze. A similar pad soaked in a 1 per cent. solution of salt must be placed on another part of the body and connected to the other pole of the battery so that the circuit may be completed. This pad, which may be termed the indifferent pad, must not be smaller in area or less thick than the other. The pads may be kept in even contact with the skin by a few turns of a bandage.

(3) The cords are made of flexible wire and insulated by means of some suitable covering. They may be enclosed in india-rubber tubing, which has the great advantage of being waterproof so that short-circuiting, caused by the contact of the accidentally wetted cords, is prevented.

(4) The source of current: The current may be taken from the mains, provided that it is a direct, not an alternating current. The voltage and amperage supplied direct by the mains are too high, and must therefore be brought down to a suitable level. This may be done by means of a switchboard with a volt-selector. When using this apparatus it is necessary to prevent the patient touching any conducting bodies which are connected to earth, such as gas- or hot-water pipes, otherwise he may receive a shock. Another apparatus, which costs more, but is safe, and, in addition, is usually fitted with a device for obtaining currents for other forms of electrical treat-

ment, is the motor transformer. For those who cannot obtain a current from the mains a portable battery of dry cells may be used. The cells will last for some months before running out, the length of time depending, of course, upon the length of usage. Or a motor transformer, worked by accumulators, may be used. A motor transformer may be obtained for working with an alternating current.

It is imperative that the current should be gradually applied, and not put on at once in full strength. If this precaution is not taken the patient will feel a shock. The current should start from zero, and be increased till it is as strong as the patient can comfortably bear. The duration of a session may be from twenty to thirty minutes, or even longer, particularly if the ions are penetrating normal skin. The sessions may be repeated every day or every other day, providing that the skin remains intact. When a session is over the current must be gradually diminished to zero before the wires are disconnected.

Blisters are likely to occur if the pads are making bad or uneven contact. Be forewarned if the patient complains of a stinging pain at one spot. If any part of the skin is not intact cover it by means of waterproof plaster.

The following chemicals have proved their value in the treatment of disease by the ionic method. Zinc ions are of great value in the treatment of chronic ulcers, and have often proved successful where all other methods have failed. They have also proved of value in the treatment of bed-sores. In the treatment of sinuses successful results can be obtained if the zinc solution can be brought into contact with each part of the wall of the sinus—often not an easy matter. A zinc rod or wire with a covering of lint or tape, soaked in a zinc sulphate solution, may be introduced into the sinus. For the treatment of moles or warts a zinc needle is made to transfix the skin at the base of the growth. It is connected with the positive pole of the current source, and a current of about one milliampère is passed for one minute. Zinc ions have been also used for the treatment of many other conditions. Reference to them will be found in a paper read by Dr. Lewis Jones at the annual meeting of the British Medical Association held at Liverpool, where a discussion on the subject was introduced by him; it will be found in the 'British Medical Journal' of August 31st, 1912. Magnesium ions have proved to act like a specific in the treatment of some kinds of warts. Lithium ions have been used in the treatment of gouty arthritis, also rheumatoid- and osteo-arthritis. In the ionic treatment of large joints the pads may be placed on opposite aspects of the joint provided they are not in contact. If many joints require treatment the conducting wires may bifurcate and conduct the current to or from many joints.

Salicyl ions have been used in the treatment of certain joint diseases, particularly osteoarthritis and rheumatoid arthritis. They have been successfully used in treating neuralgia and neuritis, such as sciatica, and of different forms of fibrositis.

Chlorine ions have proved to be very efficacious in the softening and resolution of scar-tissue such as that following burns, and that found around ankylosed joints. Dupuytren's contraction of the palmar fascia has been successfully treated by chlorine ions. Such treatment takes a long time, but the results are often surprisingly good.

The treatment of disease by the ionic method opens a wide field for therapy and research. Those who wish to study the subject cannot do better than refer to the recently published sixth edition of Dr. Lewis Jones's well-known work, 'Medical Electricity.' A book on ionic medication by the same author will be published shortly.

August 4th, 1913.

The Later Development of Prematurely Born Children.—Wall has investigated this subject with particular reference to the later development of nervous and intellectual disturbances, in a series of over 600 cases in Breslau, out of which 183 cases were subsequently traced and subjected to examination. The statement often made by neurologists, that premature children are predisposed to Little's disease, idiocy, imbecility and epilepsy, was not supported. The majority of the children developed in a perfectly normal manner, but in some there was delay in walking and speaking. This developed mental defect is not constant or irreparable. It seems as if these children merely required time to overcome the deficiency brought about by e premature delivery. The severity of the symptoms was in inverse proportion to the original weight of these children.—*American Journ. of Obst.*, lxviii, p. 143.

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INTERNAL GLANDULAR SECRETIONS AND THEIR INFLUENCE ON THE CAUSATION AND TREATMENT OF DISEASE.*

By R. MURRAY LESLIE, M.A., B.Sc., M.D.,
Senior Physician, Prince of Wales's General Hospital; Physician, Royal Hospital for Diseases of the Chest.

THE subject of internal secretions and their relation to physiological and pathological processes is one of the most important questions that can engage the attention of the modern practitioner of medicine. Sir E. A. Schäfer has recently stated that it is of quite as much importance from the clinical and pathological point of view as from the physiological, and adds that our scientific knowledge regarding internal secretions is the product of of the last twenty-five years. Even now, however, the information at our disposal is a mixture of ascertained facts (accurate knowledge) and conjectures, and it will be our task in this lecture to differentiate as far as possible between these.

The term "internal secretion" was first employed by Claude Bernard† in 1855, when referring to the glycogenic function of the liver, which, curiously enough, is not now regarded as coming precisely within the category of internal secretions. The modern theory of internal secretions may be considered in some respects as the legitimate successor of the ancient doctrine of the humoral origin of disease advocated by Hippocrates and his followers, and which obtained throughout the middle ages. It was then held as a cardinal medical principle that if a person were ill the humours of the body had somehow gone wrong. The old humoral physiology of Hippocrates and Galen referred to

* Lecture delivered at the Medical Graduates' College and Polyclinic.

† Bernard, Claude, 'Leçons de Physiol. Expert,' Paris, 1855, i, p. 96.

the various "humours," which were respectively differentiated under the terms "yellow bile," "black bile," "phlegm," etc. The humoral principle cannot be said to be fundamentally altered now that we are enabled by means of modern chemical and physiological investigation to isolate many of the specific active ingredients and to determine their actual functions in the animal economy.

It was the discovery of the marvellous controlling influence of the central nervous system on the functions of the animal tissues and organs that led to the discarding of the ancient humoral theory, but recent bio-chemical investigations have conclusively proved that the nervous system is no longer to be regarded as the only important controlling influence in respect to the co-ordination and inter-action of many of the chief physiological functions, and indicate that specific chemical reactions exert a still greater influence in this direction. Sir William Whitla, in his recent annual oration delivered to the Medical Society of London,* states that the more closely we examine the physical and vitalistic conceptions of drug-action, the more certain does it appear that behind the operation of physical laws and vital activity chemical reactions are continually taking place, which are the real cause of the physical forces being themselves set in motion, and that this is indeed a necessary consequence of the recognised laws of relationship between the chemical constitution of a drug and its physiological activity. He is of opinion that the nervous system itself is to a large extent controlled by chemical stimuli. Without going so far as the physiologist, Richet, who states that the living being is a chemical mechanism—and perhaps nothing more—many observers believe that chemical investigation will do more than anything else to explain the processes of life. Even in quite the lowest grades of animal life, before any central nervous system has come into being, we find a definite co-adaptation of function whieh can only be the result of bio-chemical activity. Thus the apparently volitional movement of amœboid organisms (*e.g.* selection and pursuit of food-particles) is undoubtedly the result of the bio-chemical principle known as chemiotaxis. Certain chemical substances, Whitla states, powerfully attract leucocytes, and their immigration into an inflammatory area is probably determined by a chemical change in the protoplasm of the injured tissue or to the presence of a soluble chemical substance in its fluid constituents. The protrusion of pseudopodia by an amœboid organism and the mutual attraction of the sexual gamete-cells may also be attributed to chemiotaxis.

Starling believes that the rudiments of the nervous system have arisen as the result of special "reactive cells" being set apart for specific purposes. Pursuing the analogy of the amœba still further, just as the presence of food-particles stimulates the chemical processes of metabolism, Bayliss and Starling have shown that the mere presence of food in the alimentary canal excites the formation of certain chemical secretions ("messenger substances"), which produce adaptation of the activities of the liver, pancreas and other digestive glands. Such co-adaptation of function is one of the principal results of the bio-chemical activity of internal secretion.

Vincent* defines internal secretion as "the preparation and setting free of certain substances of physiological utility by certain cells of a glandular type, the substances so set free being passed out into the blood-stream, either directly or indirectly by way of the lymphatics. The word 'secretion,' therefore, may be applied not only to the actual liquid or semi-liquid products formed by the glandular organs, but also to the process of their manufacture and liberation." The word "internal" serves to distinguish these products of secretory activity from "external" secretions, where the products pass into some special channel of exit, *e.g.* bile-duct or ureter. At the same time an external secreting gland, such as the liver, pancreas, or kidney, may likewise possess the function of internal secretion.

Brown-Séquard is regarded as the real founder of the modern doctrine of internal secretions. It was at a memorable meeting in Paris of the Société de Biologie in 1889 that Brown-Séquard, then seventy-two years of age, described the experiments which he carried out by means of subcutaneous injections of testicular extracts upon his own person. In his own case he found that as a result of these injections he experienced an extraordinary increase both

* Clinical Journal, June 25th, p. 177.

* 'Internal Secretions,' Edward Arnold, London, 1912.

in physical strength and mental activity. In an address before the Medical Faculty of Paris he for the first time expressed the view that "*all* animal tissues and glands with or without ducts supply to the blood substances which are either useful or essential, and the lack of which may produce disease." He showed experimentally that there is a chemical interchange between the different organs, which he regarded as of the highest importance.

By publishing his experiments in respect to the influence of the generative organs and other glands on the body generally, and the marvellous results in his own person of the administration of extracts of these glands, Brown-Séquard laid the foundation of modern organotherapy, which is even now sometimes described as the "Brown-Séquard method of treatment." It is interesting to note, however, in passing, that Celsus and Pliny used various organic substances for the relief of symptoms (*e. g.* rennet for dyspepsia, wolf's liver for hepatic disease, and stag's testicles for sexual impotency).

The recent researches in Europe and America have shown that Brown-Séquard, whose ideas were scouted in his life-time, was not only a pioneer in an entirely new department of medicine, but indicate also that his name is destined to take its place as one of the greatest in the history of medicine.

It is now held that the power of internal secretion is not a property of all animal cells but is vested in certain special cells and organs, each of which secretes a specific product which passes into the blood-stream and exerts a definite chemical influence upon other tissues and organs. Intracellular enzymes have been included by some writers among the internal secretions, even although during the life-time of the cell they are bound up in the protoplasm. Some of these go so far as to suggest that life itself may be nothing more than the sum total of the activities of the enzymes contained within living matter.

The English physiologists, Starling and Bayliss, have made a special study of gastric and intestinal secretions, and have found that the cells of the intestinal mucous membrane secrete a substance termed "secretin," which is absorbed into the blood-stream and stimulates the liver and pancreas and also the glands of the intestinal walls. To this exciting substance, or "secretin," they have given the name "*hormone*," derived from a Greek word meaning to "excite." The term "hormone" has now come to be universally used to denote all such physiological substances secreted by glands which act as chemical stimuli co-relating different organs; and all glands which produce such chemical substances capable of exciting, inhibiting or otherwise affecting the functions of other glandular organs and tissues are described as "hormone-producing" or "internal secretory organs."

As regards the mode of production of these internal secretions we know little or nothing, except that they are products of organic metabolism. In the case of organs having both "internal" and "external" secretions, these are not necessarily or even generally elaborated from the same morphological elements. Thus, in the case of the testicle, evidence is accumulating that its internal secretion is not elaborated from the spermatozoa-producing elements but from the interstitial tissues — the so-called "Leydig's cells." The semen is the external secretion, while the internal secretion is a hormone, absorbed into the blood and affecting the body, as a whole, and establishing a chemical co-relation between the different organs. The orchitic internal secretion may continue active long after the cessation of spermatogenesis.

It may be here noted that the process by which poisonous substances (*e.g.* the toxic products of proteid metabolism) are counteracted and rendered harmless is closely related to the action of these products of internal secretion, elaborated by such organs as the liver and thyroid gland.

Action of Internal Secretions.

We have already referred to the co-relating action of hermones, and have also indicated that such action may be excitory or inhibitory. It is believed by many that the inhibitory function is the predominating one, as most organs are supposed to have a superabundance of energy which requires to be kept under control. Thus defective secretion on the part of a special hormone-producing gland may be followed by hyperplasia (hypertrophy) of some particular co-related organ or tissue, due to the withdrawal of the inhibitory chemical influence, while excessive secretion may be followed by corresponding hypoplasia. This is well illustrated in the case of the ovary, atrophy of which is so often

followed by increased deposition of adipose tissue, as is so often seen in the case of women after the menopause. On the other hand, excessive functional activity of a hormone-producing gland may produce hyperplasia and hyper-function of the co-related organ or tissue, due to the excitory influence of the internal secretion. This is well illustrated by the frequency of cardiac hypertrophy in exophthalmic goitre—the so-called "goitre heart" of hyperthyroidism.

Again, a particular gland may produce both excitory and inhibitory hormones. The pituitary body may be cited as an example. Hyper-secretion of this gland may produce the hyperplastic gigantism of acromegaly, and, at the same time, a hypoplasia (atrophy) of the sexual organs, with corresponding diminution of sexual function, *e.g.* impotence or cessation of menstruation. These considerations have induced Herring to divide hormones into two groups: (1) *assimilatory*, promoting building up of living matter, and (2) *disassimilatory*, promoting decomposition of living matter. Thus if a disassimilatory hormone does not act efficiently there will be excessive assimilation and consequent organic hypertrophy.

The multiplicity of action associated with the internal secretions of particular glands is strongly suggestive of the theory of multiplicity of the hormones produced by any one gland, each having its own specific action on a particular tissue. There is the analogy of the vegetable internal secretions, such as opium, with its several independent alkaloids. The analogy of vegetable and animal elaborations may be closer than has been hitherto suspected.

As regards the chemical composition of hormones this varies enormously. Some are the result of complicated synthetic processes, *e.g.* iodothyrin (from the thyroid gland), and may be of a very complex chemical character (*cf.* adrenalin from the supra-renal gland, which is a methylamino-alcohol of pyro-catechin); others are the simple products of decomposition.

It is also interesting to note that true hormones can produce remarkable effects even in the minutest quantities. It is for this reason that organotherapy is often so effective, because we are frequently able to supply a missing hormone. A familiar instance is the use of thyroid feeding in the treatment of myxœdema.

Before leaving the question of the action of hormones it may be stated that the "concensus partium" (the co-relation of organs) is the primary function of the internal secretions. Indeed the balance of vital forces and the sympathetic relationships existing between different parts of the organism largely depend upon the integrity of the hormone-secreting ductless glands, whose main function it is to adjust the activities of the co-related organs.

As indicating the importance of internal secretions, Biedl states that it is of paramount importance in almost every department of pathology, physiology and biology. Schiefferdecker believes that the so-called trophic influence of nerves is really dependent upon the effects of internal secretion, and there are others who hold that nervous activity is itself effected by means of chemical agencies. There is probably, however, a tendency at present to rather exaggerate the importance of the *rôle* played by internal secretions, but at the same time it is undoubtedly true that certain hormones influence not only glandular cells and muscular structures, but also the cells of the central nervous system, while others, such as that secreted by the pancreas, specifically determine the general processes of metabolism.

In order to appreciate the doctrine of internal secretions it will be well to glance briefly at the various methods of investigation which have so largely added to our knowledge of their functions.

The earliest information has been obtained from *general observation*. For centuries castration has been employed for the purpose of fattening cattle for slaughter, while similar adiposity in the case of eunuchs after early castration and in women after the menopause (as the result of atrophy of the ovary) were well known to the ancients. This adiposity has recently been proved to be due to reduced metabolism of carbohydrates, consequent on the abrogated testicular and ovarian functions. Richter has estimated the reduction of metabolism after castration as amounting to 14 to 20 per cent. per kilo of the body-weight.

Probably the most important method of investigation is that of *comparison of clinical symptoms with post-mortem appearances*. A constant co-relation between these affords valuable and definite evidence in favour of the doctrine of internal secretion. In this way has been discovered the relation

of myxœdema and cretinism to atrophy of the thyroid glands, of Graves's disease to hypertrophy of the thyroid glands, of acromegaly to disease of the pituitary body, and of Addison's disease to affection of the suprarenals. In the case of myxœdema and Addison's disease there is hypoplasia of the thyroid and suprarenal substance with diminished functional activity, whereas in Graves's disease and acromegaly there is more or less hyperplasia and hyper-function of the thyroid and pituitary glands.

Operative surgery has also contributed valuable additions to our knowledge of the action of internal secretions. One might specially refer to the effects of excision of diseased ovaries, testicles and thyroid glands. Removal of the ovaries is followed by cessation of menstruation and other climacteric symptoms, which may often be entirely obviated if the surgeon is able to leave even a small portion of an ovary, which may be sufficient to maintain complete anatomical and functional activity of the genital organs. Excision of the testicles in childhood may be followed later on by conditions similar to those found in eunuchs: absence of hair on face, childish soprano voice, adiposity, and phlegmatic temperament. Excision of the thyroid and parathyroids may be followed by "cachexia strumipriva" (Kocher's) and tetany.

Experimental pathology in the case of the lower animals, including excision of glands (partial or complete) and substitution (transplantation) of glands, has yielded most valuable results and cleared up many of the true facts concerning the function of internal secretions. It is to this method that we owe the great discovery that the pancreas produces an internal secretion which plays an all-important part in carbohydrate metabolism, and the absence of which is the main causal factor in producing diabetes mellitus. It is to this experimental method also that we owe the discovery of the functions of the parathyroid glands. Above all it is to this method that we owe most of our present knowledge in regard to the intimate co-relationship which exists between the various secretory glands. If one of a pair of organs (*e.g.* the kidney) is destroyed or excised there occurs compensatory hypertrophy of the other. If one particular organ is destroyed or removed, compensatory hypertrophy (as the result of over-activity) appears primarily in the accessory organs or tissues of a similar structure. Thus, the excision of the thyroid is followed by hypertrophy of the parathyroids, so proving their functional co-relationship. On the other hand hypo-activity of any internal secretory organ may be followed by hypertrophy of organs of different structure. Thus excision of the thyroid may be followed by hypertrophy of the pituitary body. This is analogous to the cardiac hypertrophy accompanying the hypertrophied thymus of "*status lymphaticus*" or the hypertrophied thyroid of "Graves's disease," in which conditions the enlargement of the heart is supposed to be due to the continued over-production of secretory substances which influence cardiac activity.

The *experimental substitution and transplantation method* has been specially valuable from two points of view: (1) It eliminates the supposed fallacy that the resulting changes may be due to an alteration of the nerve-connections rather than to the action of internal secretions, for it is obvious that if certain characteristic symptoms and changes, which have followed excision of a gland, disappear after transplantation and reappear after a second excision of the transplanted organ, that these symptoms must be due to the influence of secretions and not to nerve influence. (2) It was the effect of transplantation of the thyroid gland in myxœdema that led to the simpler method of substitution by thyroid feeding and administration of thyroid extract, acting on the assumption that the juices of the organ would probably contain the active internal secretion. In this way a scientific basis was laid for organotherapy. Similar grafting experiments have been carried out with good results in connection with the suprarenals, testicles and ovaries.

Lastly, there is the method of *studying the actual chemical and physiological properties of extracts* made from internal secretory organs. This mode of inquiry has been prosecuted experimentally in the case of animals and clinically in man in the form of organotherapy.

We have already referred to Brown-Séquard's experiments in 1889 on the effects of testicular extracts. Just as juvenility (expressed by youthful freshness and vigour—physical and mental—glossy hair, bright eyes and clear complexion) is closely related to actively functioning genital glands, so senility (expressed by senile changes in the

skin, hair, teeth, muscular and nervous systems) bears a close relationship to diminished functional activity of the genital glands. Brown-Séquard was able to demonstrate in his own person the wonderful rejuvenating effects of testicular extracts on physical strength and mental activity, and it has recently been shown that the injection of ovarian or testicular extracts may increase metabolism by 30 to 50 per cent. in hypo-functional cases affecting the genital glands.

Stimulated by Brown-Séquard's discovery, numerous experiments have been carried out with the object of discovering the physiological activity of extracts of various other organs.

Oliver and Schäfer* in 1894 discovered the wonderful effects upon the heart and circulation produced by intra-venous injection of even minute doses of adrenal extracts obtained from the chromaphyl cells of the adrenal gland. It was found that the injection of extracts of the suprarenal and pituitary glands produced a characteristic increase of blood-pressure, while the extracts of all other organs and tissues, with the possible exception of the kidney, produced a fall in the blood-pressure. The fact that the adrenal and pituitary glands contained pressor substances, while all other organs and tissues (particularly nervous tissues) contained depressor substances, induced Livon† to classify the glands of the body into two groups—the hypertensive and the hypo-tensive—according to whether the intravenous injection caused a rise or fall in the blood-pressure. The smaller hyper-tensive group included the adrenals and the pituitary gland (and possibly also the kidney, carotid gland and spleen), while the hypo-tensive group included all the other organs—the lung, liver, pancreas, thyroid, thymus, ovary and testicle.

Schäfer and Vincent‡ found that the pituitary extracts, in addition to pressor substances, also contained in the infundibular portion a depressor substance comparable in action to the depressor substance of brain extracts. Vincent and Sheen§ believe that many other tissues and organs yield pressor as well as depressor substances. These investigators, and also Batty Shaw,|| have found a pressor substance in the kidney, and Shaw believes that there may be some relation between these pressor effects and the auto-intoxication of kidney disease.

Interesting experiments have been carried out by Starling and Miss Lane-Claypon,* which indicate that during pregnancy an influence is exerted by the developing ovum (fœtus) on the mammary glands of the mother and which acts as a stimulus to their growth. By injecting extract made from fœtuses subcutaneously into virgin rabbits a growth of mammary glands was produced, and in multiparous animals a secretion of milk. Starling and Claypon concluded that the growth of the mammary glands during pregnancy is due to the action of a specific chemical stimulus (hormone) actually produced in the fertilised ovum, the amount of this substance increasing with the growth of the fœtus and being therefore greatest during the latter part of pregnancy. It has thus been practically proved that the fœtus while *in utero* actually arranges for the provision of its own milk-supply during the first year of its extra-uterine life.

The toxic effects of subcutaneous injections of sterile organic extracts may be regarded as specific. At the same time the fact that a particular organic extract if injected subcutaneously or intravenously produces a characteristic effect, such as the raising of blood-pressure, does not by any means prove that the internal secretion of that organ acts in a similar manner in the living body.

Organotherapy will be referred to in connection with particular glands. At this stage it may simply be recorded that extracts of the thyroid, adrenals, ovary, testicle, pituitary body, pineal body, thymus, liver, pancreas, intestine, spleen, lymphatic glands and even the placenta have been used in connection with various diseased processes, but in no case as yet have the therapeutic results approximated in value to the administration of thyroid extracts, the results of which gave such an impetus to organotherapy. It must not be forgotten, however, that scientific organotherapy is yet in its infancy.

The mere fact that the injection of an organic extract produces definite effects on the organism does not of itself prove that that organ produces an internal secretion. Vincent† states that the

* 'Journ. of Physiol.,' 1895, vol. xviii, p. 230.
† 'C.R. Soc. de Biol.,' January 22nd and 29th, 1898.
‡ 'Proc. Physiol. Soc.,' 1899.
§ *Ibid.*, July 5th, 1902.
|| 'Brit. Med. Journ.,' 1906, vol. i, p. 1084.

* 'Journ. of Physiol.,' 1902, vol. xxviii.
† 'Internal Secretion and the Ductless Glands.'

following conditions should be fulfilled before the specific action of an internal secretion can be regarded as proved. An internal secretion, he says, may be suspected when the extract of the gland invariably yields a substance with a specific physiological action, but its presence can be definitely stated only when the organ concerned consists of glandular secreting cells showing histological signs of activity (granules, etc.), and when the blood which leaves the organ by its veins is found to contain the same active principles as the organ itself. He adds that to make the evidence of internal secretion complete it should be possible to recognise in the symptoms produced by extirpation the direct and reasonable effects of the absence of the active principle (internal secretion), and further to be able to remove these symptoms either by transplanting the organ in some other part of the body or by administering the active principle in some form or other. Dr. Vincent believes that the most valuable knowledge regarding internal secretions will be obtained by a careful and methodical study of clinical conditions in association with patient investigation of pathological findings, including microscopical examination of the glandular cell contents.

The remainder of this lecture will be devoted to a consideration of the internal secretions of particular organs, but it will be impossible to do more than refer to a few of the principal points, selected partly on account of their intrinsic interest and partly on account of their importance from the therapeutic aspect. Detailed information will be found in such text-books as Biedl's work on 'The Internal Secretory Organs,' and that of Swale Vincent on 'The Internal Secretion of the Ductless Glands.' The latter gives no fewer than 3000 references dealing with the subject. Batty Shaw's handbook on 'Organotherapy' is a useful compendium of information on the subject from the therapeutic aspect.

The Thyroid Gland (and Parathyroids).

The internal secretion of the thyroid is supposed to be a disassimilatory hormone affecting the metabolic processes, cardiac activity, the sympathetic nervous system and certain co-related internal secretory organs, notably the pituitary body, the thymus, the suprarenal gland and the sexual gland. It is also believed to have an antitoxic action, particularly in regard to the poisonous products of body metabolism. The colloid substance of the thyroid is a mixture of two albuminoids—the thyro-globulins. One of these—iodo-thyro-globulin—forms a third to a half of the whole weight of the dried thyroid gland and is believed to be the principal active secretion. The relationship of atrophy of the thyroid gland to myxœdema and cretinism has already been alluded to, while the marvellous effects of organotherapy in these conditions are too well known to require more than a passing reference. Apart from actual myxœdema, many other conditions, such as asthma and premature adiposity, are often the result of thyroid or parathyroid insufficiency. That Graves's disease is intimately related to abnormality of the thyroid gland can no longer be doubted, though it it has not definitely been decided whether the causal factor is a quantitatively increased secretion (hyperthyrosis), or a qualitatively altered secretion (dysthyrosis), or a combination of both. The cardiac hypertrophy can be readily explained on the theory of continued hyperfunction of the heart, due to hyperthyroidism. That there are marked metabolic changes in Graves's disease is proved by the fact that the respiratory interchange of gases shows an increase of 15 per cent. on the amount of oxygen consumed. It is interesting to note that although the symptom-complex of Graves's disease is in many respects the clinical opposite of that of myxœdema, there are certain symptoms common to both conditions, *e.g.* the falling of the hair, abnormal pigmentation, increased salivary and lachrymal secretion, changes in sexual activity, and amenorrhœa. The thyroids and parathyroids undoubtedly manufacture an internal secretion which is essential to the proper growth and normal metabolic functions of the organism, to the efficient action of the heart and to the proper development of the reproductive and nervous systems. This is borne out by a consideration of the clinical manifestations of cretinism, which are the result of arrest of thyroid function in early childhood, and which can be removed by artificially supplying the lost hormones. The thyroid gland commonly enlarges during menstruation and pregnancy, indicating its relationship to the reproductive system.

The Thymus.

The importance of the thymus gland has been

realised in connection with the well-known *status lymphaticus*. The part played by the thymus gland in this condition is unexplained. A much enlarged thymus is often found associated with a general hypoplastic constitution, *e. g.* small size of the heart, fragile structure of the bones (colloid degeneration of the thyroid gland), smallness of the external sexual organs (infantile uterus, narrow vagina, small ovaries), menstrual anomalies, height frequently above average, hyperplasia of lymphatic glands and lymphoid tissue, redness of the bone-marrow, etc. There is supposed to be a close relationship between the thymus and the reproductive organs, the function being to prevent (inhibit) premature development of the reproductive organs. Patients suffering from *status lymphaticus* are extremely sensitive to harmful disturbances of all kinds (*e. g.* tuberculosis, nephritis and so forth), and occasionally succumb to the influence of a general anæsthetic.

The Suprarenals.

The principal internal secretion of the suprarenal glands, viz. adrenalin, is elaborated by the chromaphile cells of the medullary portion. The striking action of the extracts even in the most minute doses in raising blood-pressure is one of the most remarkable phenomena known to pharmacology. During life the secretion no doubt assists in maintaining the tone of muscular and other tissues. Dixon and Brodie have localised the seat of action of adrenalin to the sympathetic terminals in the muscular fibres. The extract acts directly on the muscle-wall of arteries, and the action is quite independent of any stimulation of the vaso-motor centre. The reduction of blood-pressure in Addison's disease (due to tuberculosis and other destructive diseases of the adrenal gland) is one of the most characteristic symptoms of this malady, while the muscular asthenia may be attributed to the fact that adrenalin may be essential to carbohydrate metabolism and the metabolism of glycogen is essential to the performance of muscular function. It is stated by Loeper and Cruzon that adrenalin produces reduction of the internal secretion of the pancreas. It is also believed that the cortex of the suprarenal gland yields a hormone which exerts a special antitoxic function. The cortical secretion may be responsible for the bronzing of Addison's disease, and is also believed to influence the growth and nutrition of certain tissues and glands, particularly the organs of reproduction. Certain cases of sexual precocity in quite young children have been attributed to hyper-secretion of the cortical hormones.

As regards organotherapy, the results of the suprarenal treatment of Addison's disease have hitherto proved most disappointing, and are in no way comparable to the value of thyroid treatment in myxœdema. Richter goes so far as to say that suprarenal treatment is of no value in Addison's disease. Transplantation has, however, been carried out with some measure of success.

The Pituitary Body.

The anterior lobe of the pituitary body resembles the structure of the thyroid gland, and may have analogous functions. Both glands contain thyro-iodine. Removal of the thyroid gland is followed by increase in size of the pituitary body. It has even been called an accessory thyroid. There appears to be a physiological relationship with the thymus and supra-renals, hypo-function of the pituitary body being associated with suprarenal hypoplasia as well as with thyroid hypoplasia. There is also a close inter-relationship between the pituitary body and the reproductive organs. In pregnancy the pituitary regularly enlarges and may become hypertrophied to two or three times its normal size, and returns to normal during lactation. The enlargement is due to cessation of ovarian function during pregnancy. Atrophy of the ovary from disease is apt to be followed by pituitary hypertrophy. It has been found that experimental extirpation of the pituitary body retards the growth of young animals and also the development of sexual activity. Complete extirpation is generally soon followed by death, preceded by a definite chain of symptoms. The principal effect of the internal secretion appears to be exerted upon the processes of metabolism and development. The rapid growth so characteristic of the period of adolescence is to be partly attributed to the functional activity of the pituitary body. Hypertrophy leads to gigantism, while hypoplasia and diminished function lead to lowering of temperature, impaired sexual functions, altered nutrition of the hair and nails, and adiposity, indicating that the pituitary body exercises an important influence on fat-metabolism. Pituitary secretion also, ac-

cording to Schäfer, stimulates the mammary gland and kidney. There is also a definite action on the cardio-vascular system. Pituitary extract strengthens the action of the heart by influencing the heart muscle and cardiac nerves, and also raises the blood pressure by directly stimulating the neuro-muscular mechanism of the blood-vessels.

Schäfer has shown that the hormones which stimulate non-striped muscle are elaborated principally in the posterior lobe of the pituitary body. The relationship of acromegaly to pituitary disease is well known. Marie believed that acromegaly was the result of hypo-function, but Tambourini, Benda, Schäfer, and most modern observers believe it to be the result of hyper-secretion.

Mayer believed that acromegaly originated in some disorder of the reproductive organs, and it is certainly a matter of common observation that sexual symptoms are generally the first to appear. In women menstruation ceases, and this is followed later on by a cessation of sexual desire and atrophy of the sexual glands. Mendel is of opinion that acromegaly results from the combined hyper-function of several glands, including the thyroid and pituitary. The characteristic gigantism of acromegaly is definitely related to hyper-secretory activity of the pituitary bodies. All giants are supposed to have changes in their pituitary bodies, and Sternberg states that 40 per cent. of giants are the subjects of acromegaly. Similarly, infantilism has been attributed in certain cases to hypo-function of the pituitary body.

As regards organotherapy, the treatment of acromegaly by pituitary extract is unsatisfactory, as is only to be expected on the theory that the disease itself is the result of hypersecretion. Thyroid treatment has been followed by better results. Sir Victor Horsley has recently suggested extirpation of the pituitary as the more rational method of treatment. Pituitrin appears to have a special action in producing uterine contraction in the later stages of pregnancy owing to its specific action on non-striped muscle.

The Pineal Body.

Descartes long ago expressed the view that the pineal gland was the seat of the soul. We now know it to be an organ with an important secretion, exercising a specific influence on metabolism and nutrition, and also profoundly affecting both physical and psychical development. There appears to be a certain degree of antagonism between the pineal gland and the pituitary body. Thus, while pituitary disease leads to atrophy of the genital organs, pineal disease has the opposite effect. Destruction of the pineal gland in children under seven years of age is accompanied by physical and mental precocity. There is premature development of the genital organs, which may approximate to the adult size in quite early childhood, and there is corresponding precocious development of the sexual instinct. There is also an abnormal growth of hair and a tendency to mental precocity. It would thus seem that in early childhood the pineal gland produces an internal secretion which inhibits the development of the sexual glands. As in pituitary disease, affections of the pineal gland may be accompanied by obesity, though it is doubtful whether this fat deposit is the result of hypo- or hypersecretion. Hypo-function from disease also induces gigantism in addition to early maturity.

The Glands of the Alimentary System.

Hemmeter* states that the total extirpation of the salivary glands in the dog brings about a marked diminution in the secretion of the gastric juice, which can be increased again by giving extracts of the glands intravenously or intraperitoneally. The salivary glands, therefore, in his opinion, furnish an internal secretion which stimulates gastric secretion.

Reference has already been made to Bayliss' and Starling's discovery that a hormone called "secretin" is formed in the duodenal mucosa, which stimulates the flow of bile and pancreatic fluid into the duodenum; there is first of all a pro-secretin which is converted by the acid gastric juices into secretin. They also believe that the pancreatic secretion produces a chemical stimulus which provokes the flow of the intestinal juice in the intestinal wall.

Delezenne holds that the discharge of the bile, the pancreatic fluid and the intestinal fluid, are all effected by the same mechanism, namely, the elaboration of secretin in the intestinal mucosa

* 'Festband der Biochem. Zeitsch.,' H. J. Hamburger, Gewidmet, Berlin, 1908.

after the passage into the intestine of the acid contents of the stomach.

Edkins and others believe that there is a specific hormone in relation to gastric secretion analogous to secretin, and which might be called gastric secretin. It is believed that the first products of digestion act on the pyloric mucous membrane, and produce in this membrane a substance which is absorbed into the blood-stream and carried to all the glands of the stomach, where it acts as a specific excitant of their secretory activity.

Various secretogens are now used in dyspepsia, but it is impossible at this stage to express any opinion as to their value.

The pancreas secretes an internal secretion which prevents glycosuria by exerting a specific influence upon carbohydrate metabolism. The secretion is supposed to be situated in the islets of Langerhans, though this is denied by some. Rebendi believes that there is a functional relationship between the islets of Langerhans and the reproductive organs. There is an intimate connection between diabetes and pancreatic disease. Glycosuria constantly follows excision of the pancreas, but if even one fifth of the gland is left there is no glycosuria. Total excision of the pancreas in dogs is followed by sugar in the urine in a few hours or, at the most, a few days. Mering—the discoverer of pancreatic diabetes—believes that diabetes is due to the cessation of a specific function of the pancreas, which is essential to the normal employment of sugar in the organism. It is believed that the pancreatic hormone inhibits the formation of sugar.

The Kidney.

Sir Rose Bradford, in 1899, showed that striking changes in metabolism may be produced by the reduction of the renal parenchyma, and which have been confirmed by Biedl's experiments in removing wedge-shaped portions of the kidney in dogs, a procedure which is followed by breaking down of muscular and other tissues. There is also polyuria, the secretion of urine being two to five times the normal and accompanied by increased secretion of nitrogen.

Uræmia is now believed to be due to changes in the chemical composition of the blood brought about by suppression of the renal internal secretion. Brown-Séquard himself suggested this as long ago as 1889, and thought that uræmia might be partly explained by an absence of normal internal secretion. He found that removal of kidneys from rabbits and guinea-pigs was much more quickly followed by death than ligature of both ureters. Some authorities believe that the kidneys produce an internal secretion of pressor substances which have an effect on blood pressure.

The Generative Glands.

Reference has already been made to the discovery that the swelling of the mammæ during menstruation is due to the internal secretion of the ovaries, while the swelling of the mammæ during pregnancy when the ovary ceases to be active is due, as was first experimentally shown by Biedl, to an internal secretion or hormone produced by the fœtus itself. In proof of the fact that it is the fœtus and not the ovary that produces this internal secretion, it has been found that castration of the ovary during pregnancy does not prevent the enlargement of the mammæ. We have already alluded to the experiments of Lane-Claypon and Starling, whereby it was shown that the injection of fœtal extract into a virgin rabbit caused increased growth of the mammary glands, while similar injections into a multiparous animal caused secretion of milk.

The causation of menstruation has always been a debatable question, but the simple fact that menstruation ceases after the cessation of ovarian activity at the menopause suggested its intimate relationship with the function of the ovary. Indeed in the eighteenth century Sentima expressed the view that menstruation was dependent on the ovary.

Attention has also been drawn to the effects of castration as indicating the important *rôle* played by both testicles and ovaries, not only upon the anatomical characters and physiological activity of the genital organs themselves, but upon many other activities of the organism. Amongst other functions the internal secretions of the generative organs stimulate the processes of ossification at the epiphysial cartilages, so that late maturity tends to be accompanied by increased length of the long bones with corresponding increase in height. The relationship between juvenility and active genital organs and between senility and diminished genital functions has already been noted. The genital internal secretion increases carbohydrate meta-

bolism and hence diminished function is accompanied by fat-deposit.

Some authorities are of opinion that there is a close relationship between chlorosis and disordered genital function. Chlorosis may be the manifestation of some alteration (hypo- or hyperfunction) in the internal secretory activity of the genital glands. Even Hippocrates and Galen believed there was a close relationship between chlorosis and the functions of the ovaries. According to Wallert there is a connection between the ovarian internal secretion and the formation of blood, and Villemin regarded chlorosis as a toxic condition, owing to the internal secretion of the *corpus luteum* becoming absorbed into the blood instead of being discharged into the menstrual flow.

As supplementing Brown-Séquard's results, Zoth and Pregel in 1896 proved that the subcutaneous injection of testicular extract when combined with muscular exercises produced an increase of as much as 50 per cent. in the muscular performance as tested by ergographic records. It is believed that the internal secretion in the testicle plays a part in the genito-vesicular reflex, which prevents a simultaneous urine discharge and semen ejaculation. The secretions of the testicle and ovaries determine the principal secondary sexual characters, such as growth of the hair on the face and of the larynx in the male at puberty. The same applies to the fighting qualities of the male, as is readily proved by a comparison of the patient ox with the fiery bull, the capon with the cock, or of the phlegmatic eunuch with the Turk.

In the testicle the internal secretion is not elaborated by the generative cells, but by the interstitial cells—the so-called "Leydig's between-cells." In the case of the ovary there is a divergence of opinion, but here also the interstitial cells are believed to be the active agents, though some observers hold that important internal secretions are formed in the *corpus luteum*.

The effects of the internal secretions of the female generative organs have been summarised as follows: "The hormone produced by the ovary determines the proper development of the female organs and mammary glands, the process of menstruation, the secondary sexual characters, characteristic of femininity, and also affects the general metabolism of the body. On the other hand, the secretion of the *corpus luteum* may have a special influence on the nutrition of the pregnant uterus, and in providing a secretion which influences the fixation and development of the embryo in the first stages of pregnancy."

Both male and female generative glands (testicles and ovaries) have a close inter-relationship with other secretory organs, particularly the thyroid, thymus and pituitary and supra-renal bodies. These relationships have already been referred to in speaking of these glands.

The prostate gland is also supposed to have an internal secretion which stimulates the testicles, and prostatic extract has been used in sexual hypochondriasis, neurasthenia and other conditions.

Organotherapy has proved of great value. Orchitic extracts have valuable tonic properties for both sexes, while ovarian extracts are particularly valuable in the treatment of the various neurasthenic and psychasthenic phenomena incidental to the menopause and certain diseases of the generative organs. It has also been found useful in chlorosis and amenorrhœa. The writer has used combined ovarian and thyroid extracts with great benefit in cases of amenorrhœa in young women associated with unusual adiposity. Judicious combinations of glandular extracts are of undoubted value in neurasthenia and psychasthenia.

Relation of Internal Secretions to Tumour Formation.

A. S. F. and Helen Grünbaum * have initiated the hormone theory of cancer. These observers suggest that the excess of a particular hormone in the organism together with a lesion or irritation of the tissue may give rise to neoplastic growth. It is, indeed, conceivable that a hypersecretion acting on otherwise normal tissue might lead to the formation of neoplasm, while the relative hypersecretion resulting from insufficient absorption from an atrophied senile membrane might account for slow-growing tumours. The Grünbaums inoculated previously refractory rats with pieces of glandular organs from known susceptible organs along with sarcoma, and in two experiments the result seemed to show that the carotid gland is able to assist sarcoma growth in rats otherwise insusceptible.

Ehrlich † thinks there may be substances circu-

* 'Journ. of Path. and Bact.,' 1911, vol. xv, p. 289.

† 'Beitrage z. Exp. Pathol. u. Chemoth,' Leipzig, 1909.

lating in the organism which may stimulate the body-cells to resist the "arthreptic influence" of cancer-cells.

Askanazy * believes that certain hypoplasias of the genital organs following the formation of tumours in the ovary, testicles, or pineal body may be due to the influence of embryonal tissue formed by the tumour.

It is well known that skin moles, particularly if of some size and hairy, may become cancerous or may be present in persons who have been attacked by cancer. It is also known that there is a close relationship between ovarian atrophy and the occurrence of hair on the face of women. May it not be that the frequency of cancer in women immediately after the menopause is due to the degeneration of the ovary which we know occurs at that time, and may it not be possible that the injection or administration of ovarian extract for some time after the menopause might be of value in averting cancer? Recently the writer had under his care a case of well-marked cancer of the breast in a girl, æt. 28 years, who six months before the formation of the cancer developed a thick growth of hair on the cheek, chin, and upper lip, associated with partial amenorrhœa. Much patient clinical and experimental investigation is necessary before coming to any definite conclusions as to the existence of a definite relationship between neoplastic formation — simple or malignant — and altered function of the internal secretory organs. The subject, however, is a fascinating one, and research along such lines may be productive of results of the highest value.

August 11th, 1913.

Pulverised sugar as a dressing for cancer. —Berczeller states that the benefit from sugar applications is surprising, especially with cancer of the uterine cervix. The speculum is filled half full of "sugar dust," which is applied directly to the lesion every day or two, or three times a week. It is excellent to prepare the field for an operation or to clean up inoperable growths. The evil odours and discharge dry up at once and there is less bleeding. —*Journ. A.M.A.*, vol. lxi, p. 159.

* 'Zeitschrift f. Krebsf.,' 1910, xi, p. 397.

THREE CASES OF "FITS."

Clinical Lecture delivered in the University Clinical Medicine Theatre, Royal Infirmary, Edinburgh.

By ROBERT A. FLEMING, M.D., F.R.C.P.E.,
Assistant Physician to the Royal Infirmary, Edinburgh.

We have had in the wards three cases of "fits," each of which presented some unusual and interesting features. I propose to prefix their description by placing before you a simple classification of cerebral "fits," and then a brief account of a typical epileptic seizure in order to bring into prominence certain points of value in these three cases.

A typical epileptic fit of the *grand mal* type begins with :

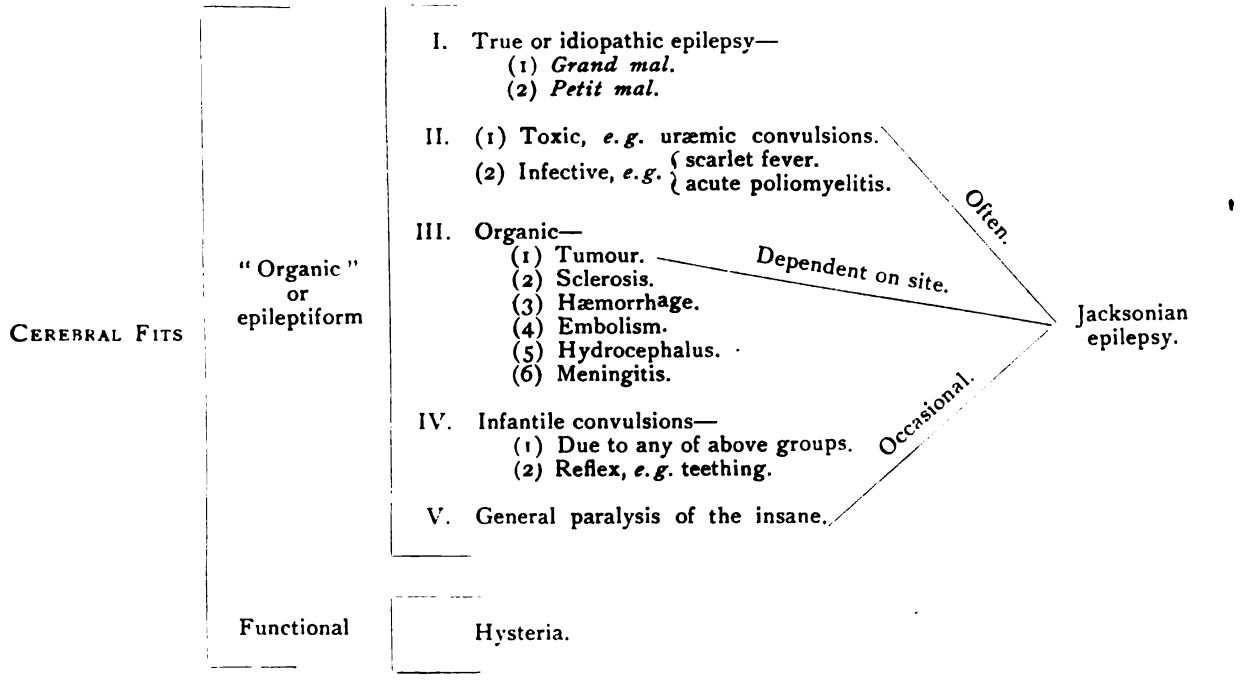
(1) *An aura.* There are many varieties of aura, but the only one to which I intend to call attention to-day is the Jacksonian type of aura—an aura which may help in the diagnosis of the site of lesion. Remember that when a Jacksonian attack becomes bilateral it leads to unconsciousness, and may constitute the motor aura of a true epileptic fit.

(2) *The cry* which immediately precedes the fit is accompanied by unconsciousness, when the patient may fall, if he either has no warning aura, or has failed to take advantage of the warning given and to place himself in a position of safety.

(3) *The stage of tonic spasm*, which lasts only for some seconds. The special clinical features of this stage to which I desire to call your attention are the dilatation of the pupils, the loss of conjunctival response, and the characteristic attitude of arms (flexion of elbows, wrists and fingers, and slight abduction of arm) and legs (extension with inversion of the feet). There is generally more spasm on one side than the other, and the head and eyes are turned towards the more convulsed side. Towards the end of this stage the patient's face becomes cyanosed.

(4) *The stage of clonic spasms*, lasting for several minutes, begins with a fine tremor, which soon becomes a coarser rhythmic movement During this stage the jaws open and shut, the tongue may be bitten between the teeth, dyeing the frothy saliva with blood, and bladder and bowels may be evacuated.

(5) *The stage of automatism* is often better



marked in a *petit mal* attack; but many epileptics after a major fit demonstrate some peculiar automatic action, such as pulling up the leg of the trousers.

(6) *The stage of stupor or sleep* which ends the attack.

After a supposed attack of *grand mal* you should verify the diagnosis by investigating the knee-jerks and plantar response. After a true fit there is for a brief time loss of knee-jerks, followed, for a variable period, by exaggeration, and co-existent with this you will find plantar extension present.

The status epilepticus is the climax of true or idiopathic epilepsy. In it there is deep coma between the fits, which are numerous, and in it the patient may die without ever regaining consciousness. As a rule the temperature runs high, and there may be hyperpyrexia. It is also of common occurrence in uræmia

A Jacksonian epileptic seizure is a sign of local irritation of a motor cortical brain area. The movements are chiefly clonic, and if they begin, let us suppose, in the thumb, they spread up the arm, gradually involving the other parts of the limb. These rhythmic movements go on for two to five minutes: there is no loss of consciousness unless the movements cross to the other side and so become bilateral; the pupils do not dilate; while the face never becomes cyanosed, although it may flush up, partly from excitement and partly from the exertion of the clonic movements of the affected limbs. Before and after the attack there is often some local anæsthesia in the affected limb, and very generally local paresis, more rarely paralysis. You will note in the scheme of fits that Jacksonian seizures are common in organic lesions of motor cortical areas in the brain, are not uncommonly present in uræmia, and are sometimes observed in general paralytics, but they are very exceptional, in fact are not mentioned in connection with functional fits.

A hysterical fit presents features entirely different from true epilepsy. There is no typical cry, no fall, but rather a graceful subsidence before an appreciative and sympathetic audience. The movements are not truly clonic, but are rather co-ordinated movements with an erratic variation, and they often lead up to opisthotonos, or sometimes the remarkable crucifixion attitude. There is no biting of the tongue, no involuntary emptying of bladder or bowels, and no dilatation of pupils, even if the patient is apparently unconscious, and there is no loss of the conjunctival reflex or abnormal change in tendon or plantar reflexes after a fit. There should, on the other hand, be some of the definite stigmata of hysteria.

With this brief description of certain of the

varieties of fits, I now show you the three cases which I desire to bring before you.

(1) The first is a boy, æt. 14 years, who was admitted to my wards on May 21st, 1913, in *status epilepticus*. He had three fits before admission and sixteen after admission, and all the nineteen occurred within twenty-four hours. He has been exposed in his occupation as a message-boy to all weathers, and five days before admission, after several severe wettings, his face was seen to be swollen, especially under the eyes; he had severe headache, and for almost the first time in his life felt seedy and unable to take his food. There is no history of any specific fever since he had measles at the age of six years. The boy was deeply comatose on admission, his skin was dry and harsh, and his temperature was 103° F. After some trouble, owing to incontinence, we got a sample of the urine, and found it contained blood, albumen, and a copious deposit of sodium urate crystals. There were blood, epithelial and hyaline casts. I saw a fit when in the ward, and noted that his head and eyes were turned to the right, and that the right side was more convulsed than the left. There was, however, no alteration in the tendon reflexes and no plantar extension after the fit. His maximum blood-pressure was 130 mm. of mercury, but his heart and palpable arteries did not suggest any permanent cardio-vascular changes. He was wildly delirious at times. The case was one of uræmic convulsions not following on a chronic nephritis or even an acute nephritis of past febrile origin, but due apparently to exposure to cold and wet.

(2) The second case is even more interesting, and in many respects much more unusual.

A boy, æt. 14 years, was admitted to my wards on May 22nd, 1913, in a semi-comatose condition. He had wakened his step-mother that morning at 3 a.m. by taking a fit, and he had a second fit as he lay on the trolley when being brought along the corridor to the ward.

His past history is instructive. He was a previous inmate of the ward, being originally sent to me by Dr. W. G. Sym on December 13th, 1912, as a case of optic neuritis which called for treatment in hospital.

He had at that time fairly typical signs of an intracranial neoplasm or a very limited meningitis. There were, in addition to the eye phenomena, severe headache, often frontal, vertigo, and cerebral vomiting, although the last was not very troublesome. There was no localising feature, unless the fact that with heels together and eyes shut he tended to fall backward and to the right, and he had a similar tendency when he tried to turn even with his eyes open, but this was not constant, and soon disappeared after some weeks stay in the ward. There was nothing abnormal as regards his reflexes or motor power, and he had no diplopia, nystagmus or anything else suggestive of a cerebellar lesion.

A von Pirquet cutaneous tuberculin test was positive. His own mother and one of his sisters had died of pulmonary tuberculosis. Further, at the apex of the right upper lobe there is evidence of an early but not very active pulmonary phthisis.

Dr. Sym, when recommending the boy for admission, wrote about his optic discs that "his optic neuritis is of descending type—not choked disc—and there is nothing definite at the macula. His vision is $\frac{6}{6}$ in right eye and $\frac{6}{12}$ in left." We examined his eyes on several occasions, and about three weeks after admission he had a few small flame-shaped hæmorrhages in both retinæ, but they soon disappeared and never recurred. His cerebro-spinal fluid was clear, contained no abnormal constituent, and did not flow under increased pressure.

The boy improved so rapidly that we sent him home, and he had been actually working as an apprentice when the present attack developed.

After admission on May 22nd he had two more fits, both at night, and neither was seen by Dr. Linzell, my house-physician, or myself. The nurse who saw the fits states that they were bilateral, but more marked on the right side, and head and eyes were turned to the right. Neither tendon reflexes nor plantar response showed anything abnormal after the fits. The pupils reacted to light and were not unduly dilated, and when I saw him in the ward about fifteen minutes after the second fit on the day of admission the conjunctival response to touch was active.

Dr. Sym's original description of the discs—and he gave the same description when he saw the patient again on his readmission—indicates that in his (Dr. Sym's) opinion the appearance of the fundus ("descending type of optic neuritis") suggested a meningitis rather than a neoplasm.

There was, however, nothing else suggestive—no irregularity of pulse or respirations, no boring of the head backwards into the pillow, and no Kernig's sign. His sight remains as previously described, and the headache so much complained of on his first admission to the infirmary is now very rarely present. There is no question that the fits in this case are of tubercular origin—whether meningitis of a remarkably limited nature or a granuloma, one cannot say; but it is remarkable that they developed so suddenly and disappeared without leaving anything definite behind. The immediate prognosis is not grave, but we have no assurance that the tubercular focus may not again give trouble. Tuberculin in any form might set up an active tubercular meningitis, while operative interference in the absence of any focal signs or symptoms is out of the question.

(3) The third case I wish to show you is of a very different type. A woman, æt. 29 years, was admitted to my female ward on May 22nd, 1913, complaining of fits occurring during the past seven weeks.

There is nothing important in the family history. She keeps house for an uncle and aunt and has fairly heavy house-work. She has been a teetotaller all her life.

In 1910 an illegitimate child was born dead (with instruments). After the confinement she lay in bed for six weeks and quite recovered. In 1912 a second stillborn illegitimate child (natural labour) was born, and in the same year the father of both children died. The patient worried much over this, and especially as the death occurred just before they were to have been married.

This year, on April 3rd, she had her first "fit" when stretching up to get something that was hanging on a door. Her right foot began to shake violently. She sat down until it passed off in about five minutes' time. The movements extended to the knee only and she never lost consciousness.

On April 7th patient sat down suddenly on the floor, a small niece having pulled away her chair. She struck her head violently against the wall and "fainted," remaining unconscious for ten minutes.

On April 14th the second fit occurred. It began in the right leg, spread up to the right arm, and lasted for five or seven minutes. The foot was "weak" afterwards, and this, the patient says, has lasted until the end of May, being worse at night.

Between April 14th and May 16th she had four more fits, the affected leg getting progressively weaker after each.

The patient is an intelligent woman, rather emotional and very depressed. She does not sleep well and admits that she is more irritable than formerly. She suffers from headaches, especially after any eye strain. Sensibility to touch and pain are slightly impaired all over the right side and most markedly in the leg. Conduction of sensation is slightly delayed. There is no hyperæsthesia, no definite area of anæsthesia and no paræsthesia. The soft palate is not anæsthetic and there are no definite hysterogenetic areas of pain. Motor abnormalities are limited to a pseudo-paresis of the right foot, and, to a less degree, of the right hand; but this passed off entirely very soon after admission to hospital. The tendon responses are active, but not abnormally so.

No other system except the integumentary calls for any reference. She has a curious eruption widely spread over trunk and limbs—sago grain-like cysts which look deep seated, but many have burst, leaving inflamed, reddish, weeping spots. The little cysts appear to be situated in the sudoriparous gland-ducts and resemble very deep-seated sudamina, and look much like a very widely distributed cheiropompholyx.

On May 25th patient had three fits, all except one being right-sided, but the last was described by one of the nurses as having involved the left side as well as the right, and also to some extent the head and face. In no fit, even this last one, which became generalised, did the patient lose consciousness.

I saw her last fit on May 27th when I was examining her eyes with the ophthalmoscope. There was slow jerking movements of right and left legs and right arm, about 50–60 per minute in rate. The fit only lasted about three or four minutes, during which the patient was able to converse, and at my suggestion she made strong efforts to control and stop the jerking movements. She states that grasping the limb tightly (the right leg below the knee) had enabled her to arrest several fits just at their onset.

There is no question that these fits are functional, although, as you note in my scheme of fits, that Jacksonian epileptic seizures are practically never functional. The interesting features are the absence

of palatal anæsthesia, hysterogenetic areas, and the usually definite glove-like anæsthesia of hysteria, while the fit simulates a Jacksonian epileptic seizure in respect of the subsequent numbness and paresis of the affected limbs.

I told the patient on May 27th, when she had her last fit, that she was not to have any more, and now, nearly seven days afterwards, she has not had another. The weakness and numbness have vanished, and she is to all intents and purposes well and able for her work.

Nothing need be said about the treatment of this last case, which was on the usual anti-hysterical lines. An interesting feature is the effect of suggestion that no more fits must or need occur.

At the time of revising the clinical lecture for printing (July 5th) all three cases are doing well. The uræmic boy has merely a trace of blood and albumen in the urine and is now able to be up daily. The boy with the fits, due to tubercular meningitis or granuloma, appears to be perfectly well, at least for the time being, and his lung condition, at no time very active, is absolutely quiescent. The hysterical woman was seen yesterday after a sojourn at the convalescent home for two weeks looking well and happy.

August 11th, 1913.

Fatal tobacco poisoning.—E. C. Garvin describes the case of a healthy girl, æt. 6½ years, who had thread-worms, and was given by her mother a rectal injection of one pint of water in which was dissolved one and a half teaspoonfuls of tobacco. The child immediately complained of faintness, inability to stand and great nausea, and soon commenced to vomit very severely. The bowels also acted, and a part at least of the injection was expelled. These symptoms continued for about fifteen minutes, then convulsions ensued and lasted about twenty minutes. The child died in collapse about forty-five minutes after administration of the injection. Fatal tobacco poisoning is rare. Tobacco infusion is an old remedy among the laity for intestinal parasites. A practitioner might, in an unguarded moment, consent to the use of this dangerous poison.—*Cleveland Med. Journ.*, p. 130.

JOSEPH BELL: AN APPRECIATION BY AN OLD FRIEND. Edinburgh: Oliphant, Anderson, & Ferrier. Demy 8vo, pp. 92. Price 3s. 6d. net.

THE late Dr. Joseph Bell, of Edinburgh, enjoyed two distinct reputations—one as a much-beloved practitioner, for long prominent in medical and academic circles, and the other (less to his liking) due to his fortuitous connection with the writings of Sir Arthur Conan Doyle. The reading public is well aware that the most striking feature in the popular detective hero, Sherlock Holmes, was suggested to his author by a notable characteristic of Bell—his power of acute observation, allied to keen intuition. The doctor was not elated by the reflected glory. "I hope," he wrote, "folk that know me see another and better side of me than what Doyle saw."

Proof of this is provided in the above excellent memoir, by Mrs. Jessie M. E. Saxby, who was privileged to know Bell in close intimacy, was the recipient of many examples of his kindness, and who, in turn, was able to render him a loving sympathy. The work, which we have perused with profit and pleasure, is strikingly unambitious in its purpose, and makes no attempt to place Bell in any technical sense, or measure his contribution to medical knowledge or education. It is avowedly an intimate record of personal relations, with the object of presenting the sterling and lovable character of the man, and, as such, it is an entirely successful and most satisfying work. By means of excerpts from letters, the reader is brought into close contact with his personality; in deftly chosen passages we have light thrown on Bell's views on religion, the professional education of the medical man, politics, literature, and such-like. It is a keen and varied mind that is shown, honest to a fault, tolerant of opposite opinions, and seasoning all his dealings with men with a spirit of kindly humour. It is a life of strenuous endeavour and well-directed charity, not least shown in the cheerful mien by which he hid his personal sorrows and lightened those of others.

We feel sure the personal friends of this most estimable man will be most grateful to Mrs. Saxby for this piece of excellent writing, so lovingly and carefully performed, and a much wider public will find in it matters of varied interest. The book is provided with eleven capital illustrations of Bell at various stages of his career, his forbears, and his home.

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AN ADDRESS

ON

GALL-STONES.*

By F. J. STEWARD, M.S.,

Surgeon, Guy's Hospital.

Gentlemen,—Gall-stones, with rare exceptions, are formed in the gall-bladder in consequence of an infective process. This infection may be by various organisms, the most usual being the *Bacillus coli communis* and *Bacillus typhosus*, which have been found within biliary calculi years after the original infection, the fluid in the gall-bladder and the cortical part of the calculi being sterile. Moreover, biliary calculi have been produced artificially by a number of observers, by infecting the gall-bladder of animals by attenuated organisms. Gall-stones, then, may be said to be a bye-product of the natural cure of infection of the gall-bladder, being the tangible result of an attempt to protect the body from the harmful effects of the entry of infective micro-organisms into the gall-bladder.

Having thus been formed, the stones may, of course, remain where they are, or may be forced into the ducts. In a vast majority of cases the former condition obtains. Post-mortem records demonstrate that gall-stones are found in the gall-bladder of a very considerable proportion of individuals dying of all causes. Moynihan says "practically 10 per cent.," his collection of post-mortem statistics working out at 9·8 per cent. On the other hand, in 11,133 post-mortem examinations made at Guy's during the twenty years ending 1911, gall-stones were found in 423 cases—that is, in only 3·8 per cent. There is thus a marked discrepancy between the two sets of figures, which is difficult of explanation. I note, however, that

* Read before the Eastbourne Medical Society.

Moynihan's figures are derived from Continental and American sources.

The condition being found so often in the absence of any evidence of local change attributable to the gall-stones, and in the absence of any definite history of symptoms pointing to gall-stones, it is commonly stated that as a general rule they do no harm, and the conclusion is a very natural one. But I question it, for the following reasons:

(1) On carefully inquiring into the clinical history of patients who have had definite biliary colic, and in whom the diagnosis has been confirmed by operation, one frequently learns of a prolonged period of disturbances of digestion which may be called *gall-stone dyspepsia*, the special features of which I shall deal with in more detail later. As an instance I may mention a case operated upon recently. Here the patient had suffered from discomfort after food, a tendency to flatulence, and occasional bouts of vomiting for some ten years before she began to have definite attacks of biliary colic.

Now, when apparently harmless gall-stones are found in the post-mortem room, how often would careful inquiry into the history, and careful examination before death, have demonstrated that the patient had indeed suffered from gall-stone dyspepsia for years, and had either ignored the undiagnosed condition or had it treated in vain. These questions are difficult to answer, but I think one can say this much—that as clinical observation is year by year becoming more careful and more skilled, especially in regard to abdominal conditions, the cases of dyspepsia caused by gall-stones that are correctly diagnosed and treated steadily increases in number.

(2) In a certain number of cases diagnosed as gastric or duodenal ulcer the operation reveals gall-stones, and nothing else to account for the symptoms.

(3) The same applies to cases of *cancer* of the gall-bladder due to gall-stones. I have performed laparotomy in some ten cases of this kind, and in nearly all I have obtained a history of long-continued symptoms of gall-stone dyspepsia. Last summer I operated upon the brother of a medical man, who for thirty years had suffered from indigestion and vague general discomfort in the upper abdomen, finally associated with rapid emaciation. Examination revealed a mass in the region of the gall-bladder, which proved on exploration to be a cancerous gall-bladder filled with stones.

(4) This leads to my next point, and that is the question of the *frequency* of this association of cancer of the gall-bladder with biliary calculi. Formerly there was some doubt as to whether the calculi stood in the relation of cause or effect as regards the cancer, but it is now generally admitted that the cancer is secondary to the gall-stones. It becomes important, therefore, to discover if possible how frequently gall-stones *do* cause cancer of the gall-bladder.

Examination of the Guy's Hospital post-mortem records for the years 1892–1911 shows that primary carcinoma of the gall-bladder was present 38 times and gall-stones 423 times. In nine of the cases of cancer of the gall-bladder no mention is made of gall-stones, so excluding these, cancer of the gall-bladder was present in 6·9 per cent. of the cases in which gall-stones were found, or in rather more than one in every fifteen cases.

It is clear, therefore, if these figures are to be relied upon—and I see no reason to doubt them—that *once* calculi have formed in the gall-bladder the danger of the subsequent development of cancer is a very real one.

Since gall-stone dyspepsia so often precedes the passage of stones into the ducts and the development of cancer of the gall-bladder, it is probable that it indicates that the stones are causing irritation. The reverse may likewise be true, namely, that in the absence of dyspeptic symptoms gall-stones may be innocuous, but if this is true, and if so how often there are no dyspeptic symptoms, is clearly a point which cannot be determined.

Apart, therefore, from the stones entering the ducts or giving rise to other complications, the foregoing considerations lead me to believe that the presence of calculi in the gall-bladder is by no means so harmless a circumstance as is generally believed. I would therefore urge the importance of recognising the condition at the earliest possible moment; for by so doing we shall not only save patients much needless discomfort, but be able to anticipate the severe suffering caused by the stones entering the ducts, and the danger of cancer or other complications. It will be obvious that this can be done only by forming a clear idea of gall-stone dyspepsia, from facts derived from a study

of the condition in the cases already mentioned, namely those of biliary colic and cancer of the gall-bladder.

The principal features of this gall-stone dyspepsia are as follows:

Pain after food is usually present, although some patients describe the feeling as one of *discomfort* rather than of pain. It is more noticeable after the evening meal at the end of the day's work than at other times, at any rate in the earlier stages, and is very commonly associated with flatulence, general feeling of abdominal distension, and at times chilliness. The pain is mostly referred to the right hypochondrium, but, as pointed out by Mayo Robson and other observers, it is at times felt more on the left side than the right, and rich, fatty food is more prone to bring it on than a plain diet.

As regards the time-relation, the symptoms appear usually about half an hour after food, but in some cases not until one and a half to two hours or later after the meal, and such cases may exactly simulate a pyloric or duodenal ulcer, even to the point of being relieved by taking more food.

Vomiting is another frequent symptom of gall-stone dyspepsia, and, as in many cases of gastric and duodenal ulcer, commonly relieves the symptoms for the time being.

On examining a patient exhibiting symptoms such as these definite evidence of gall-stones may be found. In the first place an enlarged gall-bladder may be felt, as in the case of a gentleman upon whom I operated six years ago for Dr. Richards. He had symptoms strongly pointing to duodenal ulcer, for he had typical pain several hours after food, and no doubt caused by the violent contractions of the gall-bladder upon the large hard gall-stones with which it was filled.

In the majority of cases, however, no actual tumour can be detected, but deep tenderness over the position of the gall-bladder, especially if pressure is made well under the costal margin during deep inspiration, can frequently be made out. The position of this deep tenderness is characteristic, for it is further out and higher up than that associated with ulcer of the stomach or duodenum.

On the other hand, although this tenderness is characteristic when present, it is by no means a constant sign, for tenderness may be altogether absent, or, if present, it may be in some other position. In such circumstances an erroneous diagnosis of gastric or duodenal ulcer or of appendicitis is liable to be made, for tenderness due to gall-stones may be present in such a position as to suggest each of these conditions. The following case illustrates the difficulties of such cases.

A lady was brought to me with a provisional diagnosis of gastric ulcer. She had had constant discomfort and frequent pain after food for five years, associated with a tender spot in the mid-line and one inch above the umbilicus. Two years before I saw her, her appendix had been removed, but neither this nor careful treatment before or after had relieved her symptoms. Laparotomy proved the stomach and duodenum to be normal, but the gall-bladder was found full of stones. These were removed and the gall-bladder drained, with a completely successful result.

I would therefore insist upon the importance of carefully considering the possibility of gall-stones in all cases of chronic dyspepsia, especially having regard to the fact that in every fifteen cases of gall-stones one dies of cancer of the gall-bladder.

I now pass on to the consideration of the cases in which the stones do not remain in the gall-bladder, but either enter or pass through the ducts. Here what one would expect on purely theoretical grounds is strikingly true, namely the close relation between size of stone and nature of symptoms produced by its passage or attempted passage along the ducts. The most constant symptom produced by the passage of a biliary calculus is spasmodic pain or colic, this being caused by the violent contractions of the muscular tissue in the wall of the ducts. It is clear, therefore, that the smaller the stone the less resistance there will be to its passage, and the less severe and prolonged will be the colic. In the case of very small stones the passage may be so rapid that the pain only lasts for a few minutes, and is unaccompanied by any sign of jaundice, or, in fact, any other symptom. The diagnosis of such a case may be very difficult, and the condition is usually looked upon, by the patient at any rate, as due to indigestion or abdominal cramp, or, again, seemingly severe pain occurring without any other sign may erroneously be thought to be neurotic in origin.

Four years ago a lady was referred to me for operation by Dr. Hale White. She had during more than twelve years suffered from innumerable

attacks of violent pain in the right side. The attacks commenced with the utmost suddenness, lasted from a few minutes upwards to an hour or more, and as suddenly ceased. During all this time nothing definite had been made out as to the cause of these attacks, and the patient had been rather thought to be neurotic, until fortunately she saw Dr. Hale White, who, suspecting gall-stones, advised a careful search to be made after the next attack. This resulted in finding a tiny facetted calculus. At the operation I removed some 300 similar calculi from the gall-bladder, and since this she has remained perfectly well and has had no further attacks. If the stones are somewhat larger the pain will be proportionately more intense owing to the greater violence of the contractions of the ducts. The attacks also last longer owing to the increased resistance, and as the bile-flow is obstructed during the passage of the stone a condition will be eventually arrived at when the obstruction lasts sufficiently long to cause absorption of bile into the blood-stream, with the consequent appearance of jaundice. This state of affairs was well exemplified in a patient of Dr. Shorland's upon whom I operated recently. She had suffered from attacks of biliary colic during twenty-five years, the attacks, short and sharp at first, becoming more and more severe and prolonged, and of recent years they have lasted about twenty-four hours, and have been followed by a slight degree of jaundice lasting a few days. The gall-bladder contained a large number of small facetted calculi and a few larger ones.

As the size of the stone further increases, the increased resistance to its passage results in further intensification and prolongation of the attacks with, consequently, more definite signs of obstructive jaundice, until a point will clearly be reached when the *vis-à-tergo* is not sufficient to force the stone through into the duodenum, and it becomes impacted in the common duct. In this event the characters of the attack are different, for if the stone passes through, the pain, intense though it may be while it lasts, comes to a sudden termination and the patient experiences immediate relief; whereas, if impaction occurs the pain gradually dies down and may cease altogether, owing no doubt to the duct being gradually worn out and finally ceasing to contract. At the same time the fæces and urine and the rapidly deepening jaundice point to the fact that obstruction to the flow of bile is complete. After a short interval, during which the duct dilates to some extent, and the jaundice often decreases, the attack is usually renewed in all its violence, owing probably to the duct having sufficiently recovered to resume its efforts at expulsion, and after a variable number of such exacerbations and intervals of repose, perhaps extending over several weeks, the stone may be finally expelled, with complete relief to the pain and clearing of the jaundice. If, on the other hand, the stone remains immovably fixed, the colicky pain frequently ceases altogether, although the obstructive jaundice remains, of course, complete, and is associated with other symptoms which are indicative of the secondary inflammatory changes set up.

These inflammatory changes are always present in greater or less degree when a stone reaches the common duct, and involve not only the duct itself, but the gall-bladder and neighbouring peritoneum as well, and in certain cases may spread back into the liver, causing a general cholangitis. The degree of inflammation also varies directly with the size of the stone, for I have frequently noticed when operating upon patients who had passed a number of stones that where the attacks had been quickly over and the stones small there has usually been very little in the way of adhesions; on the other hand, the cases in which one finds the gall-bladder most contracted and adhesions most marked are those in which there have been prolonged attacks of obstructive jaundice from stones of considerable size. The symptoms of this condition are *very* important, for they give useful indications as to treatment which I shall further note later on; they are—a variable amount of pyrexia, rapid pulse, pain, which may be very severe, tenderness in the region of the gall-bladder and ducts, in severe cases enlargement and tenderness of the liver, and frequently severe vomiting.

Returning now to my classification according to the size of the stone, the *next* class will include those that are too large to reach the common bile-duct at all, so that when they do leave the gall-bladder they either become fixed in the cystic duct or in the neck of the gall-bladder. In these cases the clinical features vary in an extraordinary degree, so that whereas in one case the patient becomes acutely ill with symptoms suggestive of peritonitis,

in another the condition may be only discovered accidentally. These differences are brought about by the variation in the amount of accompanying inflammatory change, presumably the result of previous infection, so that in the most severe cases a very acute form of cholecystitis, which may go on to suppuration and gangrene, is present, whilst in others the inflammatory change may be negligible. The following cases well illustrate these extremes.

A woman, æt. 53 years, was admitted into Guy's for acute abdominal symptoms that had begun quite suddenly two days previously, with intense pain, shivering and vomiting. The woman looked ill, and had a temperature of 103° F. and a pulse of 110. On examination the upper part of the abdomen was rigid and tender, especially on the right side, where a distinct tender swelling having the character of a distended gall-bladder could be made out. I diagnosed acute cholecystitis, and operated forthwith. On exposing the swelling it proved to be a tense, deeply congested gall-bladder, covered with recent lymph. This was opened after being isolated with gauze packs, and was found to contain a mixture of pus and bile and several large gall-stones. Further examination revealed another large stone firmly fixed in the commencement of the cystic duct. This was removed, a tube stitched into the gall-bladder and the rest of the wound closed. The patient made a rapid recovery.

Now note the contrast that the following case shows: The patient was sent home to see Dr. Hale White and myself by Dr. Marriott, of Hong Kong, on account of an abdominal tumour. Four months previously he experienced some vague abdominal discomfort, and consulted Dr. Marriott, who discovered the tumour. He had experienced no real pain, but admitted having had indigestion for some time previously. The tumour had increased in size, and could be easily felt by the patient. On examination, a large, tense, pear-shaped swelling was easily felt and seen in the position of the gall-bladder. It was quite painless, moved freely on respiration, and reached well below the umbilicus. He agreed to have an operation, but while the arrangements for this were being made the tumour suddenly disappeared. As this clearly proved that a stone had dropped back into the gall-bladder, Dr. Hale White and myself both adhered to our previous opinion that an operation should be performed. I accordingly explored the gall-bladder, and found it considerably enlarged and thickened and containing one large gall-stone, about 3 in. long and ¾ in. in diameter. This had clearly been for four months impacted in the neck of the gall-bladder, without producing any very definite symptoms, and had finally dropped back.

I propose now to mention a few points regarding the treatment of gall-stones.

First as to indications: I hold most strongly that there is only one rational method of treating gall-stones, and that is to remove them by operation.

This should be advised under the following circumstances:

(1) In cases exhibiting the characteristic symptoms of gall-stone dyspepsia as already described.

(2) In view of the dangerous complications that may result from gall-stones, and especially that of cancer of the gall-bladder, and in view of the smallness of the risk attaching to an exploratory operation, this should be performed in all cases of intractable chronic dyspepsia of a type strongly suggesting the presence of gall-stones.

(3) When there have been definite attacks of biliary colic, either with or without jaundice.

(4) When the gall-bladder is or has been enlarged owing to impaction of a stone in the cystic duct.

(5) In certain cases of obstructive jaundice, due to gall-stones impacted in the common duct.

To decide when to operate in these cases is, in my opinion, the most difficult point in gall-stone surgery, and requires very careful judgment, for there can be no doubt that the operation is a more serious and difficult one than a simple cholecystotomy, and the patient is usually not in a favourable condition to withstand a long operation. This being the case, it is clearly advisable in the absence of contra-indications to give the ducts a chance to get the stone through, and so finish the business. As to the length of this period of grace, that, I think, is best determined by consideration of the following circumstances. If in the presence of obstructive jaundice the attacks of pain recur from time to time, that is evidence that the stone is not completely at a standstill, but is being gradually forced along the duct and may eventually pass safely into the duodenum. This may, of

course, take some time, and there is a general opinion that three weeks should be allowed for the purpose. This period may, however, be a good deal exceeded, and I myself know one patient who had previously had several bad attacks of colic with jaundice, and after four weeks' complete obstruction still refused to submit to operation for a further week. On the last day of the fifth week he said he felt the stone pass, and in this he turned out to be quite correct.

If, on the other hand, attacks of colic—indicating contractions of the duct—are not repeated, while obstructive jaundice remains complete, I take this to be an indication that the stone is stationary, and do not wait more that about ten days if there are no indications to operate sooner.

Quite apart from the hope of the stone passing, there is a distinct advantage in waiting for this length of time before operating, for it allows the ducts to become well dilated, so rendering their examination and the necessary manipulations very much easier.

The degree of inflammatory complication is, however, the real determining factor in this question, for it is this that most seriously affects the general condition of the patient, and so may make it imperative to operate. Thus if the liver is enlarged and tender, vomiting frequent, pyrexia marked, and the general condition of the patient progressively deteriorates while the pulse-rate rises, the risk of operative interference becomes *daily* greater, and there must therefore be no further delay.

To sum up I would say: Wait a reasonable time before operating, but only if the patient's condition remains good and the temperature and pulse-rate are not rising.

I do not propose to enter into any discussion as to the relative merits of cholecystendysis, cholecystotomy and cholecystectomy for ordinary cases of gall-stones, for experience has conclusively shown that, with certain exceptions, which are steadily becoming fewer and fewer, the simplest, safest, and in all respects the best operation is cholecystotomy.

The primary object in operating for gall-stones is, of course, to find and remove the stones; but the taking of any measures which will minimise the chance of recurrence is only slightly less important. On purely *theoretical* grounds, therefore, since it is the site of the formation of the stones, the gall-bladder should be removed; but in *practice* this is done only when the gall-bladder is very seriously diseased, for it greatly increases the risk of the operation. Failing the removal of the site of origin, the next best plan must clearly be to eliminate so far as possible the *cause* of the trouble, which is infection of the gall-bladder, and this can be best accomplished by providing free unhindered drainage.

Further, in all cases of gall-stone disease secondary inflammatory changes affecting gall-bladder and ducts are present in greater or less degree, and the cure of these conditions can be brought about after the stones have been removed only by free drainage for a period more or less prolonged, according to the conditions found at the operation. Free external drainage of the biliary apparatus is therefore an *extremely* important part of every operation for gall-stones. It can be best insured by performing cholecystotomy and tying in a large-bore tube, and this, no doubt, largely explains the success, both immediate and remote, which attends this operation.

This free drainage has a further very important function, for as the current from all the different parts of the biliary apparatus sets towards the external opening, any stones that have been overlooked, or that could not be removed at the time of the operation, tend to be carried in the same direction. This fortunate result has obtained in several of my cases. In one case a stone that I had failed to remove from the hepatic duct was washed out of the gall-bladder a fortnight later, and in three others stones appeared which I had missed altogether at the time of the operation.

However carefully the operation is conducted, and however thorough the examination of the ducts, small stones, especially floating-stones in the common duct, where, be it noted, they sometimes apparently cause no symptoms at all, are liable to be overlooked. Kehr, in a recent article, lays particular stress on this point, and says that stones are left behind in either the hepatic or common duct in as many as 12 to 15 per cent. of the cases. This, I trust, is rather an over-statement, for if it is not, the stones so left certainly are not, by any means, all washed out of the wound, so that some of them must be permanently left behind. I am very much afraid that this has hap-

pened in a case I recently operated upon. Here the common duct was full of stones, and was so well dilated that I could pass my index finger down it to a point well behind the duodenum, and I felt confident that all stones had been removed. Drainage continued for three weeks, but a month later he had several slight attacks of colic, each associated with jaundice, so I fear that he has a stone in the common duct that is acting as a ball-valve. (Since this paper was written a further operation has been performed, a floating stone being found in the common duct as expected, and removed with a successful result.)

The immediate results of gall-stone operations have greatly improved during recent years, and are now extremely good. Twenty years ago, taking all cases together, the mortality was considerable, varying, according to different published lists of cases, between 6 and 20 per cent. This mortality has since been greatly reduced, and cholecystotomy, apart from the cases in which cancer is present, now has a mortality of less than 1 per cent. My own cases, excluding those of malignant disease, but including all the operations for gall-stones, wherever situated, have, I am glad to say, all safely recovered from the operation.

As to the remote results, it is difficult to give any absolutely definite information. As far as I have been able to ascertain in my own cases there have been none of actual recurrence, but two cases are not wholly successful. One I have already mentioned as having had attacks of pain and jaundice due presumably to a small stone remaining. This may have now passed, or it may require a further operation. (This, as stated, has been done.) The other patient is a lady who, some months after the operation, began to complain of slight pain and discomfort in the region of the gall-bladder, but there has been nothing at all definite. In her case the gall-bladder was somewhat contracted and did not drain well after the operation. It would, perhaps, have been wiser to have removed it.

The experience of hospital practice seems to me the best evidence that the final results of operations for gall-stones are satisfactory, for in thirteen years' hospital work I have only seen one out-patient and one in-patient for further symptoms after a previous operation for gall-stones, and as far as I can gather from others my experience is not in any way unusual.

The out-patient case appeared to me to be one of recurrence and further operation was declined. The in-patient case I will describe, as it is a good example of the exceedingly acute conditions sometimes set up by gall-stones.

A woman, æt. 40 years, and six months pregnant, was admitted last summer into Guy's Hospital with the following history: Eight years previously she had been operated upon for gall-stones, and had subsequently enjoyed good health until four days before admission, when she began to have severe pain in the abdomen, jaundice, vomiting, and shivering attacks. These symptoms had increased and she had become rapidly more seriously ill. On admission she was deeply jaundiced, her pulse was rapid and feeble, and the upper part of the abdomen rigid and tender. A few hours later she had a rigor, the temperature rose to 105° F., and she became unconscious. I then saw her with Dr. Hale White, who agreed with me that operation gave her the only chance of recovery. As she was unconscious very little anæsthetic was needed, and I rapidly cut down upon the gall-bladder. This was adherent to the abdominal wall and omentum, and contained a single stone and a small amount of bile and pus. There was also a small abscess outside the gall-bladder. As the patient's condition was so bad I rapidly removed the stone, put in a drain and sent her back to bed. The effect of the operation was almost magical, as she soon regained consciousness, temperature became normal, and rapid general improvement took place, resulting in complete recovery.

The operation mortality being so small and the risk of recurrence being so slight, you will, I think, agree with me that the facts justify the conclusion that the risk of operation for gall-stones is certainly far less than the risk of cancer of the gall-bladder if no operation is performed.

August 18th, 1913.

Multiple Intestinal Perforation in a Relapse of Typhoid Fever ('Paris méd.,' p. 490)—Pellot and Galland.—A boy, aged 15 years, on the tenth day of a relapse of typhoid developed symptoms of perforation. Laparotomy showed four perforations near the cæcum. Each was closed with a Lembert's suture and complete recovery took place.—*Brit. Jour. of Children's Diseases.*

ACUTE INTUSSUSCEPTION.*

By HUGH LETT, M.B., F.R.C.S.,
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GENTLEMEN,—As we have had an unusually large number of cases of intussusception in this ward during the last few weeks, I propose to consider, this afternoon, some of the more important clinical features of this condition.

This little patient, who was operated upon five days ago, was a typical case, and his history will serve as an introduction to the subject. He is nine months old, and, as you see, a fine, robust little boy. His mother tells us that he is breast-fed, and had never been ill until the twenty-four hours preceding his admission to the hospital.

The illness began by his screaming with pain and drawing up his legs; shortly afterwards he vomited. We are told that he screamed with pain at intervals all through the night, and that he vomited a good many times; the vomit was gastric, and not at any time bilious or fæcal. For the last seven or eight hours before admission he was "more peaceful." Blood and mucus, and nothing else, were passed by the rectum.

On admission to the hospital he looked very ill; his eyes were sunken, and he lay on his back with a listless expression; his temperature was 100·8° F. The abdomen was not distended, and on examination proved to be lax; there was no rigidity of the muscles. A lump could be felt in the epigastrium just above the umbilicus; it was elongated, with a slight curve. While the child was being examined this lump became harder, and at the same time the abdominal wall became more tense. The child cried and drew up his legs, as if he had abdominal pain. On the napkin was a small quantity of blood and mucus, without any sign of bile or fæces.

From the history of the sudden onset of the illness, the abdominal pain, the sickness, the passage of blood and mucus, and the presence of the swelling in the abdomen, there was no hesitation in making a diagnosis of acute intussusception.

Operation.—Spinal anæsthesia, stovaine. A 2-in. incision was made through the right rectus muscle. The intussusception was found in the transverse colon, and the greater part of it reduced by manipulation within the abdomen; the last three inches were brought out through the wound and reduced under the eye. Reduction presented little difficulty until the last two inches were reached. The intussusception was ileo-colic in type, and had begun at a point an inch and a half above the ileo-cæcal valve. The abdomen was closed in the following way: Two deep sutures of chromic catgut, passing through the peritoneum and all the layers of the abdominal wall, except the skin, were first inserted, and then through-and-through sutures of silkworm-gut, taking up the peritoneum. The child stood the operation very well, and towards the end actually appeared to go to sleep. Shortly after his return to the ward he had a fair-sized motion, the first part being covered by a little blood and mucus, the rest being normal. One hour after operation the child was sleeping peacefully. Subsequent progress has been uneventful.

We need not enter into a discussion on the different varieties of intussusception. It will be enough to say that they may originate either in the large intestine or small intestine, and that the majority are of the variety known as ileo-colic, the starting-point of the intussusception being in the small intestine within one foot of the ileo-cæcal valve.

Etiology.—The cause of intussusception is still obscure, in spite of many papers that have been written and much work that has been done upon this subject. Certain cases of intussusception are easy of explanation; they are those in which there is some form of neoplasm, such as a polypus or a carcinomatous outgrowth, which projects into the lumen of the gut, is seized by the segment of intestine immediately below, and moved onwards as if it were some foreign body which had to be expelled. The part of the intestinal wall to which it is attached is drawn after it, and so the intussusception is produced. But that will not explain the great majority of cases of acute intussusception, namely, those which are met with in children under one year old. In these cases we have not been able to get any further than to say that the immediate cause of the intussusception is irregular peristalsis, and that the length of the mesocolon in infants and the consequent mobility of the intestine is an important predisposing factor. The irregular

* Based on a lecture delivered at the London Hospital.

peristalsis may be caused in various ways, but it would appear that it is particularly likely to occur in children who have been improperly fed, and in support of this suggestion are the facts that the condition occurs most frequently in children who have been recently weaned, and at seasons when there is likely to be a little lapse from the regular diet owing to holiday-making (Fitzwilliams). The suggestion that irregular and violent peristalsis is responsible for intussusception is supported by the retrograde intussusception which is not uncommonly met with on the post-mortem table, the patient having died from other causes. A further point supporting this theory of irregular peristalsis is the occasional occurrence of intussusception during the acute abdominal crises which arise in Henoch's purpura.

It seems probable, then, that intussusception may be caused either by violent irregular peristalsis which results in the telescoping of one segment of the intestine into another, or by undue projection of some part of the intestinal wall into the lumen of the gut, this being seized upon, and, as it were, swallowed by the part of the intestine immediately below. This is illustrated by the intussusception met with in cases of polypus and new growth, and I strongly suspect that in other cases hypertrophy or inflammation of a Peyer's patch may cause a sufficient elevation of the wall of the intestine to produce an intussusception. In very young children an important predisposing factor is the great mobility of the intestine, due to the length of the mesentery and meso-colon.

I have had two cases in which symptoms of an intussusception developed immediately after a blow on the abdomen In both cases the intussusception began in the cæcum, and it was tempting to imagine that the blow on the abdomen caused a depression and invagination of the wall of the intestine, with subsequent formation of an intussusception.

Age and sex.—The patient whom I have shown you this afternoon is an infant æt. 9 months, and it is remarkable that no less than 70 per cent. of all cases of acute intussusceptions occur in children under the age of one year. Boys are more frequently affected than girls, in the proportion of 7 to 3.

General conditions.—It is interesting to see how frequently infants who are the subjects of intussusception are large, well-developed, vigorous, and have had little or no illness previously. Occasionally an intussusception may occur twice in the same patient. I have known of several cases in which a child was operated upon for intussusception twice within a few months, and have heard of another case in which the child was operated upon no fewer than three times for a similar condition. Sometimes another child in a family may have had an intussusception. I well remember a woman bringing her child to the London Hospital saying she thought he had an intussusception. The receiving-room officer was naturally sceptical at first, but he found the mother's diagnosis was correct. She had had another child in the hospital with intussusception some months previously, and she remembered the symptoms. Sometimes we find that there is a history of diarrhœa and vomiting immediately preceding the intussusception, but this is by no means frequent.

Facies.—The appearance of a child with intussusception is hardly less characteristic and striking than the Hippocratic facies which is seen in the late stages of general peritonitis. The child is listless, and takes but little notice of what is going on around him. He lies on his back, and every now and then a little expression of pain will pass across his face; at intervals paroxysmal attacks occur in which the child screams, and the legs are drawn up upon the abdomen. He looks ill, is pale, and the eyes are somewhat sunken. In the early stages of intussusception these characteristics are, of course, not so very marked, but as the case progresses the appearance becomes more and more characteristic.

Constipation.—It is usual in cases of intussusception in infants to find constipation at the beginning of the attack; but it is not uncommon for the child to have a motion which is stained with blood soon after the first cry of pain; after that nothing is passed by the rectum except blood-stained mucus. A very useful sign, which holds good in the majority of cases in infants, is the absence of any fæcal matter on the napkin when the condition is fully developed. This is not a reliable test in children over one year, but in a doubtful case occurring in a child less than twelve months old, any indication of fæces or bile on the napkin should make one hesitate con-

siderably before coming to a diagnosis of intussusception.

Vomiting.—Vomiting in intussusception is usual, but in many cases it only occurs after food. It is not uncommon for the mother to say that the child has brought up all the food that has been given him. Bilious and fæcal vomiting occur only in the latest stages.

In the last fifty cases which were operated upon at the London Hospital I have found that vomiting occurred in forty-five cases—that is to say, in 90 per cent. In some cases the child vomited once only, in other cases only after food.

Temperature.—The question of pyrexia in intussusception is an interesting one. It is comparatively common for the child's temperature to rise to 102°, or even 103° F., the day after operation, gradually coming down to normal during the next three or four days. But apart from this we find that many children are admitted to hospital with a temperature; that is to say, that during the attack of intussusception and before operation, the temperature may be considerably raised above normal. This is, of course, particularly interesting, as it is contrary to what we usually find in other cases of acute intestinal obstruction, when, as a rule, the temperature is subnormal.

In the above series of 50 cases I found that 32 had a temperature above normal; in 8 of these, it is true, the temperature was only 99°, but in the remaining 24 the temperature varied between 99·6° and 103°. In one case, in which the child died, the temperature was 104·6°. In 12 cases the temperature was subnormal, and in the remaining 6 it was about normal. The prognosis does not appear to be affected by the temperature, and several children with a temperature of 102° and one with a temperature of 103°, did well, as also many of the children with a subnormal temperature. From these facts we may conclude that in the majority of cases intussusception is associated with a rise of temperature, and, consequently, that in a doubtful case the presence of pyrexia does not contra-indicate this diagnosis.

Abdomen.—During the first twelve or twenty-four hours, and in some cases thirty-six hours, the abdomen appears to be quite normal on inspection; it moves well on respiration; it is neither retracted nor distended; there is no visible peristalsis. If you examine the little patient beween the attacks of pain, and can gain his confidence, so that he will lie still without crying while you examine him, you will find the muscles of the abdominal wall lax, and that there is no rigidity. I should like to emphasise this point, as I remember a house-surgeon who took me to see a case of intussusception which had just been admitted to hospital, saying, "I do not think it is a case of intussusception, as the abdomen is not rigid enough."

Abdominal tumour.—The next point, and a very important one, is the detection of a tumour in the abdomen. The frequency with which one can be felt varies greatly according to different authors, but this, I think, must be largely due to inadequate note-taking, as, when only a small number of cases are recorded by any single observer, the percentage in which a tumour can be felt is generally exceedingly high. In my own experience of forty cases, I have been able to feel the tumour in every case in children under a year old. Of course, in older children it may be missed completely if the abdomen is distended; but in infants the distension must reach an extreme degree to prevent the discovery of this tumour. A great deal depends on the way in which it is sought for. As I have already said, it is of the greatest importance to gain the confidence of the child, and to wait patiently, with the hand on the abdomen, until an interval comes in which the child ceases to cry.

The tumour is usually felt in the course of the large intestine, for the simple reason that in the great majority of cases, the intussusception has advanced as far as the colon by the time that medical aid has been summoned—if, indeed, the intussusception did not actually begin in the cæcum or colon itself. The tumour is elongated, somewhat cylindrical in outline, and frequently has a slight curve with the concavity directed towards the umbilicus. This concavity is due to traction of the mesentery on the mesenteric border of the intussusception.

There are two features of an intussusception which are of especial value in differential diagnosis: (1) While palpating the tumour it may be felt to change its consistency and become harder and more prominent. Should this be the case, the child will, at the same time, cry out with pain, draw up the legs, and the abdomen will become rigid. (2) The tumour is sometimes felt to slip

away from under the examining hand in the direction of the cæcum. This, of course, means that the intussusception has been partially reduced by the manipulation.

Another point of importance is that if, in the event of unavoidable delay, two separate examinations are made, the tumour may be found to have changed its position in the interval; for example, it may have advanced from the region of the transverse colon to the descending colon, or from the ascending colon to the transverse colon, and so on.

Rectal examination.—If a tumour cannot be found on abdominal examination, the next step is to examine the rectum; and in this connection let me remind you of the extreme value of rectal examination in all doubtful abdominal cases, whether in adults or in children. The diagnosis of many doubtful abdominal conditions has been settled in this way.

In a case of intussusception you may find, in exceptional cases, that the intussusception has actually protruded through the anus, or you may find that, although it has not passed so far, it has entered the rectum and its apex can be felt with the tip of the finger. It is sometimes described as feeling like the os and cervix uteri. You may however, still be unable to feel any tumour, and in that case, keeping the index finger of the right hand in the rectum, place the left hand upon the abdomen. In this way it is possible to examine bimanually a great deal of the abdominal cavity in an infant, and so discover a tumour which otherwise would have been missed.

Differential diagnosis.—In a typical case of intussusception, such as the patient I have shown to you this afternoon, there should be no doubt as to the diagnosis, above all when a tumour can be felt in addition to the other signs, namely, the passage of blood and mucus by the rectum, the paroxysmal attacks of abdominal pain, vomiting, the general appearance of the child and the rise of temperature. Gastro-enteritis and colitis are sometimes mistaken for intussusception, but the appearance of the child is different; there is no tumour to be felt, and if the child is passing blood and mucus by the rectum, there is practically always a certain amount of fæcal material or bile to be seen on the napkin, apart from the different history in the two cases.

Henoch's purpura, again, may give rise to difficulty in diagnosis, for in the abdominal crises in this condition the pain is very severe, the patient vomits, and blood and mucus are passed by the rectum. But, as I have pointed out in another place,* Henoch's purpura is rarely met with in children under three, and in many of the cases there are other evidences of Henoch's purpura, such as the purpuric eruption on the body or limbs, a history of joint pains a short time previously, hæmaturia, and so on. It is true that intussusception may supervene upon an attack of Henoch's purpura, but the risks of operation are so grave in Henoch's purpura from sloughing of the wound with secondary peritonitis, hæmorrhage, and so on, that unless a tumour with characteristics conforming to those of a tumour caused by intussusception can be felt in the abdomen, leaving no doubt as to the presence of an intussusception, operation should not be performed.

Appendix abscess.—I have known a case of appendix abscess to be mistaken for an intussusception; but in these cases the duration of the illness, the septic appearance of the patient, the tenderness of the swelling, its fixity, and the rigidity of the abdominal muscles over it help to differentiate between the two, and further, examination of the rectum may reveal the presence of a tender, œdematous, and perhaps fluctuating swelling projecting into it.

Operation.—In treatment by operation there are two vital essentials; firstly, that the condition should be relieved as soon as possible after its diagnosis, for every additional hour that the intussusception remains unreduced makes the prognosis worse; and secondly, the utmost precautions must be taken to minimise shock. The little patient is already suffering from a certain amount of shock as a direct consequence of the intussusception; a laparotomy, with the necessary manipulation, still further adds to this. It is therefore important to operate as rapidly as possible and so diminish the time during which the patient will suffer from the shock of operation, and, secondly, to prevent this shock as far as possible. These two desiderata can be fulfilled by operating under spinal anæsthesia, in a way which is impossible with a general anæsthetic. In the first place, as has been abun-

* 'Rep. Soc. Study Dis. Children,' vol. viii, p. 307.

dantly proved, operation under spinal anæsthesia is associated with far less shock than operation under general anæsthesia. Secondly, the abdominal conditions when the patient is under spinal anæsthesia are ideal from the point of view of the operator. The abdominal walls are lax, there is no straining, and there is little tendency for the intestines to prolapse through the wound, so that the operation becomes a very easy one, and can be rapidly executed. The actual time of operation varies from five to ten minutes. After the operation there is no sickness, and the child can be fed at once—a most important matter in the case of infants.

The incision which I usually employ is a vertical one through the middle of the right rectus muscle, about the level of the umbilicus. Through this incision one can easily reach the parts of the intestine which are most frequently the seat of intussusception. The index and middle fingers of the right hand are inserted into the abdomen and the intussusception reduced as far as possible. When no further reduction can be effected within the abdomen, the intussusception is brought out through the wound, and by compression of the intussusception and gentle pulling on the entering loop the intussusception is completely reduced. If this is not possible, and the intussusception is irreducible, it is necessary either to resect it or perform a lateral anastomosis. Of the two, the latter is preferable in infants under one year, as resection is attended by a terribly high mortality.

By means of two buried chromic catgut sutures I include the peritoneum with the two layers of the rectus sheath and the rectus muscle itself. I then insert three silkworm gut sutures, passing through the skin and all the other structures of the abdominal wall. These are removed at the end of a fortnight, and the wound is then supported by a broad band of strapping, which is passed round the abdomen and left on for another week. A valuable point in the after-treatment is the administration of saline by the rectum. In the case of an infant it is improbable that more than two or three ounces will be retained at a time, but if the child can absorb this amount it is of material advantage.

Treatment by injection. — Some time ago there were many who maintained that it was unnecessary to operate upon children with intussusception, as the intussusception could be reduced by injecting water or milk into the large intestine under pressure or by inflation with hydrogen. At first sight this seems to be reasonable, and to possess distinct advantages over laparotomy, and it is true that in a certain number of cases this method of treatment has been successful. But that it cannot be successful in all cases is obvious when we consider the time and patience which are sometimes necessary to effect the reduction of the last inch of the intussusception, and when, with the greatest care, little rents will appear in the peritoneal coat before this reduction is effected; and lastly, there are those cases which cannot be reduced, and have to be treated either by resection or lateral anastomosis.

We must take it, then, that a certain proportion of the cases cannot be reduced by injection. Further, we have to face another disadvantage, namely, that if the last part of the intussusception, which is often so difficult to reduce at operation, remains unreduced, it may not be detected on abdominal examination, and so may result in an early recurrence of the symptoms. Again, if reduction is incomplete, and it becomes necessary to operate after all, not only has valuable time been lost, but also the patient is suffering from a certain amount of shock from the manipulation which he has already undergone.

We must conclude, therefore, that inflation or injection should only be employed in exceptional cases.

Spontaneous recovery.—This may take place in one of two ways— by sloughing of the intussusception or by spontaneous reduction.

A very few cases have been recorded where a child who has suffered from intussusception, and for some reason or other was not operated upon, has passed a large slough *per rectum*, which was evidently the intussusception, which had undergone gangrene and become separated off from the rest of the intestine, but this is extremely rare.

That spontaneous reduction does take place sometimes is beyond all doubt, but it is a rare occurrence, and it is beyond human nature not to be a little sceptical when laparotomy has been performed, and the surgeon says that the intussusception which he felt before operation has been reduced spontaneously. But the appearance of the intestine, which was recently the seat of the intussusception, is sufficiently characteristic, the peritoneal coat is injected, and that part of the wall of the intestine

which formed the apex of the intussusception is thickened and slightly depressed.

There are many other points which I might have entered into in discussing intussusception, notably, multiple intussusceptions, retrograde intussusception, and so on; but my object this afternoon has been to try to impress upon you the chief clinical features of this condition, and to help you to bear in mind the essential points in its diagnosis and treatment.

August 18th, 1913.

Poisoning by Odourless Illuminating Gas.—E. Thomsen describes the following incident. A party was given in winter in a one-storied house with ten rooms. On the following night all the twelve persons sleeping in the house suffered from intractable vomiting, violent headache and lassitude. As none of the guests who slept out of the house was affected, and three persons who slept in the basement of the house and did not share in the festivities were included among the sufferers, the food and drink of the party were clearly not to blame. The patients recovered during the day, but on the second night their condition was similar to, but even worse than on the first night. They recovered as soon as they slept elsewhere, and three volunteers who slept in the house developed the same symptoms.

Poisoning by noxious fumes was suspected, but their source was not at first evident, for the house was not supplied with gas. During the previous summer repairs had been carried out on the drains. A new water supply and soil pipe had been laid on, and an old cesspool had been closed. The frozen ground over this was now explored, when gas escaped which was odourless and inflammable. This was, therefore, clearly not sulphuretted hydrogen. Besides, toxic symptoms recurred among the inmates of the house after the old cesspool had been thoroughly cleaned out. The mystery was solved when, several yards from the house, a leak was detected in a gas pipe as it passed over a water pipe. The escaping gas was deodorised by the earth through which it filtered, and its course was through the old cesspool, and up a ventilation shaft which opened under the house, and thus enabled the gas to invade all the rooms.—*Petersburger Med. Zeit.*, p. 30.

CIRRHOSIS OF THE LIVER.

By J. MACKIE WHYTE, M.D.,
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ALTHOUGH excessive drinking is prevalent in Dundee, cirrhosis of the liver is not very common; indeed its comparative rarity in Scotland has long been known. The presumption that cirrhosis is caused by a combination of factors, amongst which alcohol is not necessarily one, is borne out by a study of the two following cases.

CASE I.—*Cirrhosis of liver; much enlarged spleen; repeated attacks of hæmatemesis during three years, of which the last proved fatal. Rupture of varicose œsophageal vein found at autopsy.*

Alexander B—, æt. 27 years, of irregular and unsatisfactory habits. Inclined to take drink to excess since he was nineteen. He said beer was his usual tipple; his mother, however, said it was whisky. Had been at sea for three years; afterwards a mill-worker. Did little work during the last three and a half years of life.

June, 1908: *First attack* of hæmatemesis, lasting two days. In bed for three weeks, during which he vomited blood once more.

January 6th, 1910: First admission to infirmary. *Second attack* of hæmatemesis began yesterday. Just before tea, sudden irrepressible desire to vomit. In a few hours, vomited four times "four to five pints." Walked to infirmary, where he had been attending as out-patient for ulceration about toes. No dyspeptic symptoms. On January 8th vomited two pints of blood. No pain before it, but some epigastric pain after. Melæna; stools not free from blood for another week. Anæmia severe: red corpuscles = 925,000, hæmoglobin = 46 per cent.; no poikilocytes or erythroblasts; leucocytes of normal ratio. Liver dulness, 2½ inches in nipple line, nowhere absolute; left lobe not to be mapped out at all. Spleen much enlarged, movable, firm, and smooth, measuring 11 in. in length. Stomach normal to examination. Improvement speedy, successive counts showing rapid formation of blood, the red corpuscles being 3,000,000 on March 6th.

July 15th, 1910: Second admission to infirmary. No stomach troubles, but a painful

swelling at left wrist which soon went away; also a more severe, very tender swelling, affecting outer side of right ankle and the parts above and below. My colleague, Mr. Greig, diagnosed it as osteitis with cellulitis. With rest, Scott's dressing, massage, movement under chloroform, and bandaging, he recovered slowly, and was discharged in November. It should be noted he took the anæsthetic badly, being very excited in the second stage. Red corpuscles = 4,500,000. Spleen and liver as before; no ascites now or at any time.

March 10th, 1911: Third admission. *Third attack* of hæmatemesis. after fourteen months of freedom. Only two or three mouthfuls vomited, but probably a good deal of concealed hæmorrhage. Red corpuscles = 2,400,000 hæmoglobin = 65 per cent. Sent to convalescent home in a month.

September 1st, 1911: Final admission. *Fourth attack* of hæmatemesis began the day before. Several pints of blood vomited between October 31st and September 2nd, when he died. A good deal of epigastric pain after the hæmorrhage began. Professor MacEwan was consulted as to advisability of operation on day of admission, but decided against it. Various other measures, such as rectal salines and venous transfusion of saline fluid, were tried, with no great benefit. Much restlessness. No rise of temperature during any of his attacks.

Post-mortem examination by my resident, Dr. A. R. Moodie, and myself. Stomach and duodenum showed no ulceration. At lower end of œsophagus, several well-marked varicose veins, one of which was filled with a dark thrombus and showed an ulcer the size of a millet-seed. Mucous membrane of stomach grey, with numerous black spots. Stomach and intestines full of blood-clots. Liver reduced in size, dense, weight 50 oz. Left lobe showed long, flattened projection behind. Quadrate and Spigelian lobes unusually prominent. Surface rough, but capsule shiny, showing no sign of perihepatitis. Projections on surface very irregular in size. On section lobulated, pale, and fatty-looking. Spleen 9 × 8 in.; weight, 24 oz.; elliptical, flattened, fairly firm, but not preserving its shape; easily indented with finger. Small greenish-yellow infarct on inner surface at upper end. Kidneys fatty.

CASE 2.—*Cirrhosis of liver in non-alcoholic subject; ascites when* 8½; *laparotomy; development retarded; slow toxæmia, with rapid development of symptoms at end.*

Elizabeth F—, æt. 18 years, admitted to Dundee Royal Infirmary on September 29th, 1911. Complaints: Pain in left side for a week; breathlessness and cough for an uncertain time. She was the eldest of a family of twelve, of whom six survive, the others having died in infancy. Father said to have hip-joint disease, and there is other evidence of tuberculosis in family, but not of syphilis. Patient brought up on cow's milk, on account of maternal mastitis. Had whooping-cough, measles, and intestinal catarrh. Never got alcohol. When 8¾ years old was in infirmary under Professor Stalker, and was transferred to care of Mr. Greig on account of ascites. For nine months there had been observed a gradually increasing abdominal swelling, with restlessness at night and loss of appetite. She was delicate-looking and small, but fairly nourished. No pain, no anæmia or diarrhœa; tongue clean. No definite evidence of tuberculosis. Abdomen 28½ in. at umbilicus; liver enlarged—lower border 2 in. below costal margin. Mr. Greig operated on December 16th, 1901, when circumference was 31 in., 2½ in. of a gain in three weeks. Bowels examined through incision—no signs of tubercle; no enlarged glands, spleen normal, liver large and firm, rather rough on surface. The fluid was serous. Recovery excellent without rise of temperature. Some fluid reaccumulated, but when she left, on February 8th, circumference was 20½ in. After this she seems to have kept fairly well, though backward, and by no means strong and healthy. When she came to the allowed age, she worked in a mill for a year or two, but her mother had to take her home, as she was unfit for this. She was slow at house-work, and inclined to drowsiness. In October, 1910, March, 1911, and September, 1911, she was in the infirmary, under the observation of my colleague, Prof. C. R. Marshall, and myself. She was languid, lacking in mental and physical energy. Eyes prominent, subject to Koch-Weeks' bacillary conjunctivitis (Dr. McGillivray). Voice hoarse, nails clubbed, abdomen tumid (30 in.), with no dulness in flanks. Had an explosive cough. In both March and September complained of a not very severe pain in epigastrium, or right hypchondrium, or præcordia.

No jaundice or cyanosis. Appetite poor, bowels constipated. Liver enlarged, at various times from one to three finger-breadths below costal margin, well felt in mid-line, halfway between xiphoid and umbilicus with firm, smooth edge. Spleen enlarged forwards and downwards, easily palpable. Costal margins bulged. A systolic pulmonary murmur, a venous pulse in neck. Pulse in October generally 96 to 116, sometimes slower (64 to 80). While note at lung apices was not quite satisfactory, nor breath-sounds quite right, nothing indicating active disease was found. No tubercle bacilli in sputum. Temperature subnormal. Had never menstruated. Blood examination on at least three occasions showed nothing distinctive. On the last, red corpuscles = 4,800,000. Hæmoglobin = 95 per cent. Leucocytes = 3200. The X rays in March showed nothing wrong in heart or lungs.

She remained in this condition, in bed and not asking up, till October 20th, when temperature rose to 101·8° F., pulse to 108. Evening temperature, 103°. Next day became delirious in morning. Breathing became deep and laboured, rather like air-hunger; she had a dazed look; pupils dilated, reacting to light; arms and legs very stiff; speech indistinct; she repeated any word she used several times. She was cyanosed. No sugar or albumen either now or previously in urine. Knee-jerks became greatly exaggerated; ankle clonus and extensor response present. At 2.30 p.m. dyspnœa very great; not relieved by transfusion of sod. bicarb. or O_2 inhalation. Temperature kept up, and she died on October 22nd.

Summary of Prof. Sutherland's *post-mortem* report: Fairly well nourished. Short scar in mid-line below umbilicus. Pericardium contained 2 oz. of brownish fluid. Heart practically normal. Lungs slightly adherent, deeply congested. Occasional caseous nodules found towards apex of left lung. Bronchial glands normal. Bronchi congested; contained muco-pus. Spleen weighed 18 oz.; soft, deeply congested. Kidneys large, congested. Stomach somewhat distended; shows patches of congestion, with numerous hæmorrhages. Similar patches in intestine, especially its lower part. Liver weighed 45¾ oz.; surface markedly irregular, especially over convexity. Right lobe more reduced than left. Appearance suggests a coarse, multilobular cirrhosis with fatty change. Gall-bladder distended, with black-brown bile. Ovaries showed menstruation had not begun. Surface-vessels of brain somewhat engorged, but no definite change was found in brain substance.

In both of these cases, in spite of advanced cirrhosis, we note the *absence of ascites* at time of death. In the man it seems never to have existed, and in such a case death from hæmorrhage is more likely. The collateral circulation was well developed at the junction of stomach and œsophagus, and probably elsewhere: so that much of the portal blood got into the general circulation without traversing the liver. But the œsophageal varices are not an improvement on nature. They are a standing menace, being badly supported, and exposed to trauma by swallowing hot or otherwise hurtful food or drink. As in all varicose veins, thrombosis is apt to occur, and after that ulceration comes easily. In the girl, ascites was present for months ten years before her death. She was lucky to come so well through her operation, for even the simpler method of puncture is apt to be followed by death at no distant date. Rolleston mentions the case of three children—two sisters and a brother—all operated on by laparotomy for what turned out to be cirrhotic ascites. Two had been supposed to have peritoneal tuberculosis, and the third a perforated typhoid ulcer. They all died soon after the operation. The result in Elizabeth's case is not so wonderful as the cure of abdominal tuberculosis by incision, but there is some analogy. The collateral circulation must have been relieved of pressure sufficiently to prevent much fresh effusion, and to establish fresh routes for the blood. Absence of ascites in pure cirrhosis is noted *post-mortem* in 30 per cent. (Rolleston) to 56 per cent. (Kelynack and Fenn).

The *age and causation* in those two cases are interesting. In the girl, alcohol must be excluded; there is no reason to suspect congenital syphilis, which is said, on good authority, to be sometimes responsible for multilobular cirrhosis. Intestinal catarrh may have had something to do with it, and one must bear in mind the tubercular tendency of the family and of the girl herself. In the beginning of 1912 I had under my care a badly developed little girl of 10, who had a large, hard liver, reaching 4 in. below ribs, and almost certainly cirrhotic, with a large spleen. There was no evidence of syphilis, but there was of tuber-

culosis; the mother had died of consumption, and there was dulness at the apex of the right lung. She is in much the same state yet, but has recently had a suppurating cervical gland. Lardaceous disease can be excluded for certain. It is said that over 600 cases of cirrhosis of liver in children have now been recorded; so it is no rarity. In the man's case, the ulceration between his toes and the joint affections were evidence almost conclusive of syphilis; and this, plus alcohol, would account for the liver condition at his early age better than alcohol by itself, especially as he seems not to have been a tippler so much as a periodically excessive drinker.

While there was an absence of ordinary indications of dyspepsia, the general nutrition and health were affected in both. The languor, drowsiness, and backward development of the girl were, no doubt, due to slow toxæmia, and it is not unlikely that the man's disinclination for work during his last years was of physical rather than moral significance. Whence came the rapid development of toxic symptoms in the girl at the *finale*? She was living on the edge of a precipice, like subjects of nephritis and diabetes. In the so-called cholæmia, though jaundice happens to be present, the bile-element seems to be of secondary import; death is essentially due to the same causes as in this case. Some failure of kidney excretion, some excess of poison production, or the simple summation of stimuli, must be invoked as the solution. The liver is now regarded as ever on the watch against toxins absorbed from the alimentary tract. Adami has shown it has also a bactericidal power.

August 18th, 1913.

Treatment of Sleeplessness.—Moszeik describes a method which, he says, seldom fails. On going to bed assume a comfortable attitude in which every muscle is relaxed, but not the attitude in which you are accustomed to go to sleep, but something resembling it. Every movement, coughing, yawning, are strictly repressed, especially the desire to turn over. The same attitude is maintained, constantly resisting the longing to move or turn over. As a rule, by the end of fifteen or twenty minutes, you grow very drowsy and then, just as the desire to turn over becomes irresistible, you turn with the least possible effort and assume the position in which you habitually go to sleep, and sleep follows at once.—*Journ. A.M.A.*, vol. lxi, p. 316.

FIRST STEPS TOWARDS A STATE MEDICAL SERVICE. By BENJAMIN MOORE, D.Sc., F.R.S., M.R.C.S., L.R.C.P., President of the State Medical Service Association. An address delivered before the Medico-Legal Society of London, January 21st, 1913. Pp. 28. Published by the State Medical Service Association, 1913. Price 6*d*.

PROF. MOORE gives three main reasons for the institution of a State Medical Service—national efficiency and physique, monetary saving, and scientific ideals. His plan would embrace in its workings every member of the profession, to whom he holds out the prospect of an average salary of £500 a year with a retiring pension at sixty-five, on similar lines to the higher branches of the Civil Service. Since 55 per cent. of all deaths are caused by infectious diseases, the cause of which is known, organised preventive medicine is the main objective, and for the rest the cost is shown to be, in the end, a saving upon the services at present in operation. It is urged that the health service should be under the administrative control of a cabinet minister possessing professional and technical knowledge, and under whose jurisdiction the permanent staffs of the various health offices of the Local Government Board, Board of Education, Home Office, Boards of Agriculture and Fisheries and Insurance Commission would be placed, so that overlapping would be avoided and greater efficiency obtained. Since the whole profession would be included it is suggested that the difficult problem of vested interests might be overcome by fixing each practitioner's income on a basis of the past three years' receipts from his practice, with a retiring pension *in lieu* of purchase value.

Glowing, indeed, are the utopian prospects held out, such as abolition of hospital abuse, book-keeping and bad debts, with certainty of definite annual holidays, and daily hours off duty, time for post-graduate study, etc. But, as in similar schemes, the fact seems to be lost sight of that we have to deal, not with the standardised parts of some huge machine, but with an infinitely complex society in which the personal equation, diversity of individuality, establishment of confidence between doctor and patient are things that matter intensely when dealing with disease in its prevention and cure. However, the address is well worth reading as an able statement of the case for a State medical service.

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THE CAUSES AND TREATMENT OF GLYCOSURIA.

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GLYCOSURIA is the result of an increase in the quantity of dextrose in the blood. In consequence of this hyperglycæmia, a certain amount of sugar passes through the kidney into the urine. Phloridzin diabetes is the only exception to this, the glycosuria being caused by the action of phloridzin in the kidney, hypoglycæmia resulting. Phloridzin diabetes thus differs entirely from all other forms of diabetes. Probably the renal cells are rendered more permeable to blood sugar, although some experimental evidence has been held to show that the dextrose is split off from serum proteins. Phloridzin diabetes will not be considered further.

Normal urine has a reducing power which corresponds to an amount of dextrose of 0·2 per cent. to 0·5 per cent. Special methods have to be employed for this estimation. This reducing power is due to various substances. Isomaltose is a constant constituent of urine, but only in traces. Uric acid and kreatinin also are reducing agents. But the amount of these substances present in urine is insufficient to account for the total effect. It appears that urochrome, the normal pigment of urine, is responsible for the greater part of the reduction. In spite of a large amount of work it is still undecided whether or not normal urine contains any dextrose at all. Probably it does not. In any case the amount cannot exceed a minute trace.

Dextrose is always present in blood. The accurate method devised by Rona shows that the amount is about ·08 per cent. This is considerably less than was formerly believed.

Organs concerned in the Production of Hyperglycæmia and Glycosuria.

(1) Kidneys.

It might at first sight be supposed that glycosuria is due to increased permeability of the kidneys to sugar. It is undoubted that renal disturbances cannot account for the phenomena of diabetes, human or experimental, since hyperglycæmia is invariably present. In the later stages of chronic diabetes the kidneys become less permeable to sugar, but this is of little importance.

(2) The Liver.

The liver is the sugar factory of the body. Other organs can influence and control this power of the liver, and other tissues consume sugar, but the liver alone produces it. The carbohydrates absorbed from the intestine reach the liver, are there changed into and stored as glycogen, and this is converted into dextrose, and passes as such into the circulation for utilisation by the tissues. These steps are generally accepted, although Pavy held other views.

The liver retains this power over carbohydrates even when diseased. An advanced cirrhotic liver shows a slight diminution of its power of dealing with carbohydrates, especially in the case of lævulose, but it is only in extreme conditions, such as phosphorus poisoning and acute yellow atrophy, that the glycogenic function is seriously impaired.

Dextrose of the blood is mainly used by the muscles, and when these are working hard the consumption is greatly increased. The amount of dextrose in the blood remains constant during ordinary muscular work, and is only slightly diminished by prolonged exertion. Therefore the output of dextrose from the liver must vary with the demand. In some manner the requirements of the muscles are conveyed to the liver, and lead to an increased production of sugar. How this is done has not yet been discovered. This passage of the carbohydrates from the intestine to the liver, and from the liver to the tissues, may be regarded as the normal course of their metabolism.

In diabetes, natural and experimental, no glycogen is present in the liver, and the amount of sugar in the blood is greatly increased. Obviously the sugar factory of the liver is disturbed. These two facts can be accounted for in the following ways:

Firstly, the liver may be unable to form glycogen. On this hypothesis carbohydrates pass direct from the alimentary canal into the general circulation. Naunyn considered that this was the essential cause of diabetes. On this view the liver is primarily to blame.

Secondly, the glycogen, though formed, may be rapidly converted into dextrose. It becomes merely a transient intermediate product between the absorbed carbohydrates and the blood sugar. This might result from a permanent increase in the call for sugar from the tissues.

There is little evidence on which we can arrive at any decision. Both factors may be present. In any case the fact that the sugar functions of the liver are disturbed does not prove that this is the primary cause of diabetes, since it may be the result of abnormal stimuli arising from other organs. Some of these stimuli will be considered below. There is little doubt that the original fault does not lie with the liver.

(3) The Pancreas.

It was long ago observed that the pancreas was frequently found to be diseased in diabetes. For many years all attempts to produce glycosuria in animals by removal of the pancreas were unsuccessful. We now know that this was due to incomplete extirpation.

Disease of the pancreas, such as neoplasm, may be very far advanced without glycosuria being present. On the other hand, in diabetes it may be difficult to find any abnormal condition in the pancreas, but von Hansemann has lately brought forward evidence that changes are invariably present.

There are several theoretical ways by which the pancreas may affect the carbohydrate metabolism. The influence of the external secretion can be excluded experimentally by leaving a portion of the pancreas separate from the intestine. No pancreatic juice reaches the intestine, and yet there is no glycosuria until this remnant is removed. The action of the pancreas must therefore be internal. This might be due to the destruction of some substance circulating in the blood, the presence of which tends to cause increased sugar

production by the liver. The importance of this view will be seen when we consider the influence of adrenalin. On the other hand, the pancreas may be excreting some substance into the circulation, the absence of which results in glycosuria. This hormone may either have a controlling influence on the production of sugar by the liver, or may be a necessary condition for the utilisation of sugar by the tissues. This latter possibility will be referred to again. Whichever explanation is accepted, it is undoubted that the pancreas has an internal secretion which controls the amount of sugar in the blood directly or indirectly, and the absence of which leads to hyperglycæmia.

(4) The Suprarenal Glands.

The injection of adrenalin causes hyperglycæmia and glycosuria. Thus the internal secretion of the suprarenal has an effect on the carbohydrate metabolism which is the opposite to that of the pancreas. Adrenalin has this power even in starving animals, whose liver has been emptied of glycogen by the administration of phloridzin, the sugar in these circumstances being produced from protein. Repeated doses of adrenalin lose this power of producing hyperglycæmia. Possibly this is due to a "compensatory response" on the part of the pancreas. The action of adrenalin is especially marked in de-pancreatised animals, and in diabetes. On the other hand, in Addison's disease, the percentage of dextrose in the blood falls, and this also occurs in animals in which the suprarenals have been extirpated.

The suprarenals and the pancreas may thus be looked upon as controlling in opposite directions the sugar factory of the liver. It has been suggested that diabetes is due to adrenalin acting without the control of the pancreas. The experimental evidence is at present inconclusive. The double operation of removal of pancreas and suprarenals cannot be done satisfactorily owing to the collapsed state of the animals.

(5) The Nervous System.

It is undoubted that the nervous system has a control over the sugar functions of the liver. Claud Bernard's famous experiment of puncture of the floor of the fourth ventricle proves this. Cerebral tumours, accidents to the head, stimulation of certain nerves, and even psychical disturbances may lead to hyperglycæmia and glycosuria. This hyperglycæmia ceases when the liver is glycogen-free, and the sugar disappears from the urine. It is not impossible to suppose that repeated stimuli of this nature would produce a long-continued glycosuria. This has been held to exist, and has been termed "neurogenous diabetes." In such a case the glycosuria would be independent of diet. Formerly the diagnosis of neurogenous diabetes was often made on this ground in young people with diabetes, but it is now realised that the glycosuria in such cases is frequently unaffected by diet or only influenced by prolonged treatment.

The nerve impulses may in theory act on the liver directly, or indirectly through influence on another organ. H. Meyer has shown that the nerve impulses pass through the left splanchnic nerve to the left suprarenal, and hence to the right suprarenal. The right splanchnic nerve apparently takes no part. The stimulation of the left splanchnic produces an increase in the amount of adrenalin in the blood and causes marked hyperglycæmia. Hence it has been argued that the nervous system acts on the sugar factory of the liver entirely by this means. If this is so, then it should be impossible to produce glycosuria by stimulation of the splanchnic in animals from which the suprarenals had been removed. This has been proved to be so. A few experiments in this connection are still difficult to explain, but the balance of evidence strongly favours the view that the nervous system acts on the liver indirectly by causing a secretion of adrenalin into the circulation.

(6) Thyroid Gland.

The administration of thyroid extract, in large doses, leads to glycosuria. It has long been observed that glycosuria is common in Graves's disease and the assimilation limit for carbohydrates is low. On the other hand, in myxœdema the assimilation limit is high. The experimental evidence is complicated by the difficulty of removing the thyroid gland without injury to the parathyroids. The clinical evidence seems to prove definitely that the internal secretion of the thyroid results in an increased production of sugar by the liver. It is possible that the parathyroids have an opposite effect.

(7) Pituitary Gland.

It has been noticed that glycosuria is very common in acromegaly, suggesting an action of the pituitary gland on the carbohydrate metabolism. In agreement with this, injections of the posterior lobe (*pars nervosa*) of the pituitary gland lead to hyperglycæmia and sometimes to glycosuria, while if the lobe be removed the assimilation limit becomes high. The two lobes of the gland are physiologically and developmentally distinct. The secretion of the posterior lobe passes into the cerebro-spinal fluid while the anterior lobe is a ductless gland.

The two lobes of the gland may have different effects, but the experimental evidence is at present incomplete. It has lately been observed that in advanced acromegaly the assimilation limit for carbohydrates is increased, and Cushing has suggested that the glycosuria of early acromegaly is due to irritation of the posterior lobe, and that atrophy subsequently occurs. In acromegaly there is increased glandular activity. When the secretion is diminished, the subject shows adiposity and sexual infantilism (dystrophia adiposo-genitalis). In this condition the assimilation limit for carbohydrates is very high. Glycosuria often occurs in tumours of the pituitary gland.

There is no doubt that the pituitary gland has some control over the carbohydrate metabolism, but of the method of action little is known.

The Varieties and Origin of Diabetes.

It is generally agreed that the term "diabetes" should be applied only to cases in which dextrose is present in the urine over a prolonged period. It is practically impossible to make the urine of a healthy man glycosuric by giving him starch, but it is always possible to effect this by giving a large amount of sugar. The definition excludes such an incident as this, in which one may believe, for convenience, that the liver is unable to deal with all the carbohydrates presented to it, and that some sugar escapes into the general circulation and is excreted by the kidney in consequence of the hyperglycæmia.

Have all forms of chronic glycosuria a common basis? If so, this must be loss of pancreatic function, since the pancreas is undoubtedly diseased in a large proportion of the cases. In acromegaly and in Graves's disease chronic glycosuria is common. It is not impossible that these diseases disturb the interworking of a system of the ductless glands. That these glands do work together in many instances can scarcely be doubted. The pancreatic secretion may be interfered with in this manner. There are, however, no indications that such cases should be treated as diabetes.

There is a form of chronic glycosuria which is sometimes referred to as "hepatic glycosuria" or "alimentary diabetes." It occurs in patients about fifty years of age, and shows little tendency to progress. The subjects are often stout. Probably these are mild forms of pancreatic diabetes. They should be treated as such, with a leaning to mercy. There should be periods of strict diet and the carbohydrates must be watched and regulated, but a considerable amount and variety may be allowed.

If the pancreas is at fault, does the disturbance always lie in the cells of the Isles of Langerhans? Some years ago Dale tried to upset this old theory. He produced evidence to show that these cells do not differ physiologically from the rest of the pancreas. His views are not generally accepted. Histologically there seems to be a difference in these cells in cases of diabetes, but the appearances in the stained sections are difficult to interpret and are still the subject of discussion.

How does disturbance of the pancreatic function lead to hyperglycæmia? Firstly, the adrenalin in the blood may be free to act unopposed on the liver. There is no evidence to decide this point. Secondly, the pancreatic secretion may act directly on the liver; or thirdly, it may act on the tissues, preventing the destruction of dextrose by them.

O. Cohnheim published some experiments to prove that if extract of pancreas and extract of muscle be added to blood, this became powerfully glycolytic, although neither extract alone had this effect. The results were probably due to the action of micro-organisms. More recently Knowlton and Starling have shown that if the blood of a depancreatised animal be perfused through its heart the destruction of dextrose is very small, but if a carefully prepared extract of pancreas be added the consumption becomes normal.

If repetition confirms this series of experiments, they will have proved that in diabetes the primary error lies in the inability of the tissues to oxidise

dextrose in the absence of pancreatic secretion. Accepting this view, it may be supposed that the tissues are unable to distinguish between dextrose which they cannot utilise and dextrose which does not exist, and consequently that the stimulus passes from them to the liver, which calls forth an increased production of sugar. The normal existence of such a stimulus may be deduced from the response of the liver to the increased demands of the muscles during hard work. Such a theory is intelligible, and explains more of the known facts than any other. It may be put forward as the best working hypothesis available.

Finally, the question may be asked, Why does the administration of carbohydrates in diabetes lead to increase in the hyperglycæmia? It might be supposed that since the tissues need dextrose, the absorption of large quantities from the alimentary canal would benefit the patient. It is well known to have the reverse effect.

It may be suggested that the result is not solely due to the condition of diabetes. The liver deals with carbohydrates as they come from the portal system. It may be accepted that the greater the quantity of carbohydrates, the more actively will the sugar functions of the liver set to work. In the irritable condition of these functions in diabetes this response becomes pathological and continues after withdrawal of the stimulus.

Treatment of Diabetes.

Rollo was the first to observe that glycosuria could be controlled by the exclusion of sugar and starch from the diet, and he noticed the clinical improvement which followed this. The restriction of carbohydrates is still the sole effective treatment of diabetes, and the only improvements which have been made are efforts to elaborate this method. Glycosuria is merely the outward sign of hyperglycæmia, and it is this which must really be treated, although for clinical purposes the estimation of the sugar in the blood is impracticable.

The hyperglycæmia persists for some time after the urine has become free of sugar. In treatment it is therefore of the greatest importance to get rid of the last trace of sugar, for until this has been effected hyperglycæmia will still be present. The blood will not become normal until the urine has been free from sugar for a period of at least a fortnight, and often of several weeks. A trace of sugar is often allowed to remain for fear of acetonuria occurring if the diet is made too strict. As we shall see later, these are not the cases in which acidosis is a danger.

The reduction of the hyperglycæmia is of great importance in order to avoid the complications which are so plentiful in diabetes. It is well known that it is in the less severe cases that these complications are most often seen. Such patients may excrete merely a trace of sugar, but the important fact is that they suffer from a long-continued hyperglycæmia.

There is a further reason for bringing the blood to a normal state. In diabetes the sugar functions of the liver are in an excitable state, and carbohydrates are its most powerful irritant. Hence it is reasonable to apply to the liver the principles of rest, and then to bring it back to its normal work by graduated increases. This view may be accepted as a working hypothesis. An overdose of carbohydrates not only leads to an increased excretion of sugar, but this increase persists even when the carbohydrates have been discontinued. Obviously the overdose has left the liver in a weaker and more irritable condition.

For these two reasons it is extremely important that the urine should become sugar-free. Every effort should be made to remove the last trace. When this has been accomplished, the diet should then be persisted in for at least two weeks before any attempt is made to add carbohydrates. If a patient on a strict diet, who still has a trace of sugar in the urine, is given a more liberal diet, he will improve temporarily and feel better, but it must be understood that such treatment is incorrect and will be paid for in the future.

We will first consider the various diets which may be used in the treatment of diabetes, then the special difficulties caused by acetonuria, and lastly the treatment of a few types of glycosuria.

Diets.

In a severe case of diabetes any change of circumstances may induce the onset of acidosis.

In no case of diabetes should the carbohydrates be suddenly withdrawn. The reduction should always be gradual. When a case first comes under observation, a diet should be given resembling as closely as possible that to which the patient has

been accustomed. The excretion of sugar should be noted, and then the reduction of carbohydrates carefully commenced.

There are three degrees of carbohydrate-free diet:

(1) *Strict diet.*—This consists of meat, eggs, fish, green vegetables, bacon, and butter, and diabetic bread may be given.

This should also be used as the standard diet to which carbohydrates are added when the assimilation limit is being ascertained. When carbohydrates are being withdrawn, the patient should be given this diet with the addition of bread, the amount of which can be diminished gradually.

(2) *Vegetable diet.*—Consisting of bacon, butter, eggs, and green vegetables. The amount of fat allowed in these diets should be very liberal.

(3) *Fast days*, on which weak tea or whiskey and soda is given.

No restriction of fluid should be made. It should be remembered that not only the total amount of sugar but even the percentage very often rises with the quantity of water passed. Ordinary milk is not allowable, but a sugar-free milk has been prepared by Dr. Hutchison and is sold by Messrs. Callard.

Oatmeal Cure.

For convenience this will be described here. Von Noorden observed that cases with acetonuria frequently improved when given oatmeal. He has never claimed that this is a cure for diabetes; it is only a method of controlling acetonuria. He gives the following rules for its use:

(1) First diminish the glycosuria as far as possible by a strict diet.

(2) Give a "vegetable" diet for three days previous to the oatmeal.

(3) Give 250 grm. of oatmeal a day, for not more than three days. The oatmeal can be combined with vegetable diet, but must never be given with meat.

(4) A vegetable day should follow the oatmeal, and then two or three days of "strict diet."

The oatmeal treatment should only be used occasionally, not oftener than once in three weeks. It is of no use in mild cases of diabetes, and is solely useful for the control of acetonuria. In severe cases the preliminary diet has to be relaxed, but a vegetable day should always follow.

Acetonuria complicating the Treatment.

The terms "acidosis" and "acetonuria" should not be used as synonyms. Acidosis should be applied to cases in which toxic symptoms are present. Acetonuria refers to the fact that acetone bodies are present in the urine. The occurrence of acidosis is the great difficulty in the treatment of diabetes. If carbohydrates are removed too suddenly from the diet, acidosis and coma may follow. But the avoidance of acetonuria is often carried to an extreme. Patients are permitted to excrete a large amount of sugar rather than a trace of acetone. If a slight ferric chloride reaction develops while the carbohydrates are being diminished in a mild case, sugar is often added immediately to the diet, and thus the only effective treatment abandoned. We shall see that this is a mistake.

Acetonuria is revealed by the presence in the urine of "acetone" bodies, commonly acetone, aceto-acetic acid and β-oxybutyric acid. The origin of these bodies is not yet decided. It is certain that they do not arise from carbohydrates, whilst it is very easy to show by chemical formulæ that they can be derived from fats. Now, none of the bodies mentioned have a toxic action, but it has been found that β-amidobutyric acid can produce coma. Further, in some extreme forms of acidosis, leucin and tyrosin are found in the urine. It is difficult to see how these latter bodies can be derived from fat, but they are among the regular disintegration products of protein.

For these reasons we believe that protein is the main source of acetone bodies, but it is possible that the fats also contribute.

Let us consider in what circumstances acetonuria arises. It is recognised that acetone bodies are produced when the liver is devoid of glycogen. If the diet of a healthy man be suddenly changed to one containing no carbohydrates, acetonuria develops. Now the normal man differs from a diabetic in the fact that his liver has the power of retaining glycogen. If the carbohydrate-free diet be maintained the liver will in time form glycogen from protein, and the acetonuria disappears. This has been proved by experiment. Carbohydrates in the diet cannot be essential to the metabolism of the liver, since the inhabitants of the Polar regions eat very little and carnivorous animals none at all.

In these conditions the liver must be exercising its power of forming glycogen from protein. Possibly it is always doing so to some extent.

It may be noticed that children, who suffer so frequently from acetonuria, have a special liking for sugar. It would seem that all children suffer in some degree from "carbohydrate starvation," and that their fondness of sweets is physiological.

Undoubtedly the special factor which renders a diabetic patient so liable to develop acetonuria, is the absence of glycogen from the liver.

We will now refer to the manner in which acetonuria complicates the treatment in some typical forms of diabetes.

(*a*) Mild Cases of Diabetes.

In such cases, when they come under observation for the first time, the glycosuria diminishes and finally disappears as the carbohydrates are reduced. Originally the ferric chloride reaction is negative, but it not uncommonly happens that acetone bodies appear on the restricted diet. It is in such cases that the mistake is often made of returning at once to carbohydrates. The acetonuria is physiological, and would occur in healthy people on a similar diet. The restriction of carbohydrates should be maintained, when the acetonuria will be found to diminish, until merely a trace is present, and this will disappear completely when carbohydrates are recommended.

There is a group of cases in which a slight degree of acetonuria is originally present and persists, although the glycosuria responds to treatment, and does not return on a moderate carbohydrate diet. The behaviour of the glycosuria corresponds to mild cases of glycosuria, yet the prognosis in these cases is bad. The urine, however, should be kept free of sugar, with periods of strict treatment, and occasional days of vegetable diet, and oatmeal cure. Alkalies should be given by the mouth from time to time. Complications are especially common in this type. The prognosis in this group is specially bad when the patient is young. In middle life the prognosis is better, but the complications are troublesome.

(*b*) Moderate Cases.

We may take as such cases those in which the urine becomes free of sugar only when the protein is reduced, in addition to the complete withdrawal of carbohydrates. Acetonuria may or may not be present. The difficulty in these cases is that as the carbohydrates are being diminished, and whilst there is still glycosuria, acetonuria may develop and continue to increase. The treatment should be persisted with, and alkalies given from time to time, while a careful outlook is kept for acidosis. The earliest symptoms of this are headache, epigastric pain, sleeplessness, and loss of appetite. On the occurrence of these the diet must be relaxed and alkalies given in large quantities. On the disappearance of the symptoms treatment of the glycosuria should be resumed.

These cases are especially suitable for the oatmeal cure, periods of which should be given. The treatment of these cases may be very prolonged. In some instances the acidosis returns on every attempt to reduce the carbohydrates, and finally this has to be abandoned. Occasionally patient treatment converts the case into the type described as mild.

Usually the result lies between these extremes. Careful treatment reduces both the glycosuria and acetonuria to a point at which neither is dangerous. In such a case the sequence of treatment may be as follows:

Strict diet and bread, 80 grm.—4 days.
Strict diet—3 days.
Vegetable diet—3 days.
Oatmeal diet—3 days.
Vegetable diet—2 days.
Strict diet—3 days.
Vegetable diet—2 days.
Oatmeal diet—3 days.
Vegetable diet—2 days.
Strict diet—3 days.
Strict diet and bread, 40 grm.—3 days.
Vegetable diet—1 day.
Strict diet and bread, 60 grm.

By such means the glycosuria can be coaxed down while the acetonuria is prevented from reaching a dangerous level. On discharge the patient is allowed a certain amount of carbohydrates, with occasional stricter periods. For example:

Strict diet and bread, 80 grm.—6 days.
Vegetable diet—1 day.

Every two months a period of oatmeal cure is given, while once every year a month should be spent under observation. In carrying out such a

scheme the subject usually has greater patience than the prescriber, but it is more essential for the latter.

(c) Severe Cases of Acetonuria.

When acetonuria is advanced, any reduction of carbohydrates may lead to symptoms of acidosis, and little can be done. The treatment to be followed will be discussed under severe cases of glycosuria.

Treatment of Mild Cases of Glycosuria.

In many patients glycosuria disappears when no carbohydrates are given. Such patients are usually about fifty years of age, and in them the prognosis is comparatively good.

It should be made a rule that the slighter the case the stricter the diet, since the fear of acidosis is not present, and permanent improvement may be expected.

The diet should be gradually reduced, until a strict carbohydrate-free diet is being given. This should be done in all cases, even when the urine becomes free from sugar before carbohydrates have been entirely discontinued. During this period the hyperglycæmia is being reduced. After a fortnight of such diet, bread may be added, giving at first two ounces a day, and gradually increasing the amount. Before each increase is made there should be a day of a carbohydrate-free diet. The increase may be made every fourth day. When sugar appears in the urine carbohydrates should be reduced until this again disappears. We have now found the assimilation limits of the patient. It is general to allow daily two thirds of the maximum amount on which the urine is sugar-free.

Ordinary life can then be resumed. The diet prescribed will be a strict one together with this amount of carbohydrates, whilst once a week a "vegetable" day is interposed. In this manner, with occasional periods of stricter supervision, the patient may live for many years.

Severe Cases of Diabetes.

A case of diabetes must be regarded as severe when, on a moderate carbohydrate diet, the amount of sugar excreted exceeds the intake. Some of this sugar must be formed in the liver from protein. In such a case the glycosuria still remains when the proteins are diminished and the diet is carbohydrate-free.

Now it is a matter of little importance to the organism whether 20 grm. or 60 grm. of dextrose is excreted daily, and it is better for the patient to excrete 60 grm. on a sufficient diet than 20 grm. on one which is both insufficient and ineffective. Hence these cases should not be kept permanently on a carbohydrate-free diet.

The treatment, however, should commence with an attempt to reduce the glycosuria as far as possible. We may regard this as resting the liver. The various degrees of diet described above should be employed. The patient is changed from a full diet to a strict diet to which 100 grm. of carbohydrates are added. Six ounces of bread contain about this. The amount is gradually reduced until a strict sugar-free diet is being taken. If acetonuria develops, a course of oatmeal is given. In any case two or three days of vegetable diet should be given and occasionally a fast day, the strict diet being continued in the intervals. By these methods the glycosuria is usually considerably reduced. Carbohydrates are then added gradually to the diet. It has been suggested that the optimum amount of carbohydrates is shown at the figure where the difference between the intake and the excretion in the urine is greatest. At this point there is the maximum destruction of carbohydrates in the body. As a general rule these cases may be allowed 80–100 grm. of carbohydrates daily.

When the patient resumes his ordinary life, he should have this amount of carbohydrates added to a strict diet. The carbohydrates are best given as bread, as patients feel the absence of this very greatly. Occasional carbohydrate-free days should be ordered, and, depending on the severity of the case, vegetable days and even fast days (best spent in bed) should be arranged for now and then. A scheme should be drawn up. The following is an instance, covering three weeks:

First week— 6 days strict diet + 100 grm. bread.
1 day strict diet.

Second week—6 days strict diet + 100 grm. bread.
1 day vegetable diet.

Third week— 5 days strict diet + 100 grm. bread.
1 day vegetable diet.
1 day fasting.

These patients should be given a period of treatment under regular supervision once a year.

Between these two types, the mild and severe, come a number of transitional cases of all degrees of severity. The treatment of these must be based on these principles, modified as necessary.

Treatment of Coma.

When coma threatens, alkalies should be given in large quantities. By the mouth doses of 100 gr. may be taken. Continuous enemata, containing 3 per cent. of sodium bicarbonate, can be injected to a total of 40 to 50 grm.

When coma is present, a sterilised solution containing 0·8 per cent. of sodium chloride and 4 per cent. of sodium bicarbonate may be injected intravenously. Injections of solution of dextrose have been given intravenously, but only very transient benefit can be expected from this.

Drugs.

The only drug of any value in the treatment of glycosuria is opium. This seems to have an effect in removing the last trace of sugar, and may be employed at such a stage. Codeine has no advantage over opium.

August 25th, 1913.

Diaphragm of trachea after tracheotomy.—K. Justi.—A girl, æt. 7 years, a month after tracheotomy for laryngeal diphtheria, began to suffer again from dyspnœa. A second operation was performed at the site of the old wound, when a sickle-shaped band involving the whole lumen of the trachea, but thickest on the dorsal aspect, was seen at the upper end of the tracheal wound. Three months later the band had become transformed into a membrane and was removed by polypus forceps, when it was found to consist of connective tissue. It was certainly not caused by the tracheotomy tube, but probably by the cicatrisation of a diphtheritic loss of substance. Nine months after the first tracheotomy the child was finally able to do without a tube and complete recovery resulted. Only three similar cases have been recorded.—*Brit. Journ. Child. Dis.*, vol. x, p. 367.

ADENOIDS AND ENLARGED TONSILS.

By T. MUIRHEAD MARTIN, M.D., C.M.,
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While working in the nose, ear and throat department of a large hospital I have been struck by the preponderance of patients merely complaining of nasal obstruction. In a very large proportion, especially in children, the peculiar condition of development known as the "adenoid facies" is seen. Again, in the examination of children in a school clinic I have found nasal obstruction of sufficient degree to be noted in at least 70 per cent. Though nasal obstruction may be caused by other conditions, *e. g.* by polypi, by deflected septum, enlarged turbinates, foreign bodies, etc., the majority of cases in children are caused by adenoids.

The following table of age incidence is culled from 700 cases operated upon at the Prince of Wales's Hospital, Tottenham, for enlarged tonsils and adenoids during the ten months ending December, 1912. The number of cases is small but instructive:

Age.	Per cent.	Age.	Per cent.
1	·71	14	5·1
2	3	15	3·59
3	6	16	1·7
4	8·3	17	1·8
5	10·7	18	·5
6	13	19	·4
7	9·4	20	·4
8	6·1	21	·3
9	6·2	22	·3
10	5	23	·2
11	5	24	·2
12	6·7	25	·1
13	5·6		

These figures represent only the ages at which the patients came under treatment. They show that adenoids and enlarged tonsils sufficient to cause marked inconvenience are most frequently met with between the ages of four and seven years, and they are very constant up to the age of fourteen years. About and after puberty, the naso-pharyn-

geal cavity becoming relatively larger, sharing the facial development, obstruction to respiration is consequently less likely to be found. Still, those figures may be taken as indicative of the age-periods when adenoids are largest and most troublesome. The inspection of school-children, by the establishment throughout the country of school clinics for the treatment of these conditions, has given an increased interest to the subject of adenoids and enlarged tonsils.

The term "adenoids" includes such terms as "adenoid vegetation," "post-nasal growths," "naso-pharyngeal growths" and "hypertrophy of the pharyngeal tonsil," and means a collection of lymphoid tissue in the naso-pharynx, which may be directly or indirectly the cause of certain symptoms and conditions. This tissue occurs in the middle line of the naso-pharynx, occupying the vault, the fornix and posterior wall, and also extending, continuously or interruptedly, to the lateral walls as isolated patches in the naso-pharyngeal grooves on the margins of the mouths of the Eustachian tubes and even the nasal septum; certain masses of lymphoid tissue form a large central cushion-like mass —the pharyngeal tonsil—or such tissue may be arranged in diffuse or longitudinal ridges, which under certain circumstances becomes hypertrophic, giving rise to the condition usually designated as "adenoid growth."

The function of this lymphoid tissue is stated by some to be in part protective, to aid in excretion when the system is charged with poisonous substances, and to form a bulwark against septic invasion. Dr. Hugh T. Ashby ('Brit. Med. Journ.,' May 31st), says that children with enlarged tonsils and adenoids have a diminution of lymphocytes in the blood, but an increase in the total number of leucocytes. For this reason he says "it looks as though the enlargement of the tonsils and adenoids were an attempt on the part of Nature to supply the deficiency in the other lymphoid tissue of the body." Others hold that, like the appendix, the existence of this tissue is apparently purposeless. Dr. Ashby has examined the naso-pharynx of a number of fœtuses, and has found adenoid tissue in every one after the age of six months. The tendency of this tissue is, sooner or later, to atrophy; but why hypertrophy should exist in some cases and not in others is still a matter of controversy.

Ætiology.

(*a*) A marked predisposing cause of "adenoids" is the smallness of the naso-pharynx. Many do not appreciate how small a cavity the naso-pharynx is, even in the adult. Its height is estimated to be 20 mm. and its antero-posterior diameter from the hard palate backwards is 20–22 mm. In the infant it is much smaller, about 6–7 mm. and 9 mm. respectively. It is less of a vestibule and more of a narrow passage running obliquely backwards and downwards from the constricted opening of the posterior nares. The soft palate of the child seems to be placed more horizontally than in the adult, and bounds its anterior position below. Its shape is so peculiar that measurements fail to give the proper idea. But imagine the openings of the posterior nares on either side large enough to admit the end of a medium-sized male catheter, and you can conceive how congestion of the nasal mucus in infancy, plus the consequent mucous secretion, may close the opening from the nose to the pharynx.

(*b*) The naso-pharyngeal function of the young subject is very important, rhinitis is very common, and the purulent change which takes place in the secretion from its stagnation (due to inability of the child to expel it) is doubtless an exciting cause of adenoid growths. The infection of the mucous membrane of the nose causes inflammatory swelling, and, with the presence of mucus, interferes with the air-entry through the nose, the infective process in the nose setting up a similar condition in the naso-pharynx; or the trouble starts in the pharyngeal tonsil, the nasal trouble being secondary.

(*c*) There is a hereditary tendency in families to a superabundance of lymphoid tissue; the strumous type, and a deepening of that lymphatic diathesis in which one finds a great tendency to fibrosis in various organs of the body, notably in the nasal organs and throughout the respiratory tract. The factors producing this state are not yet understood.

(*d*) Among the children of the poor adenoids are remarkably frequent, giving rise to the theory that they are due to either poor nutrition (and possibly this in turn signifies rickets, starvation, early tuberculosis), or overloading of the system with effete products, thereby causing flabby, relaxed tissues. They are almost always due to unhealthy con-

ditions existing during the first three years of life.

(*e*) Rickets, rheumatism, and various exanthemata—*e. g.* measles, pertussis, scarlet fever—lower the resistance of the tissues generally, and are characterised by a tendency to inflammation of adenoid tissue.

(*f*) Other causes are irritation of the nose and throat by foul air in ill-ventilated rooms.

(*g*) Thumb-sucking, the use of comforters, and pap-feeding are causes, in that the circulation of blood and lymph in the tissues of the mouth and pharynx and naso-pharynx are not sufficiently stimulated.

Symptoms.

Those vary in degree. The more immediate effects of nasal obstruction are noticed in the nasal catarrh, the mouth-breathing, and the consequent depressed mentality of the child from lack of air. There is chronic nasal catarrh and liability to acute inflammation of the middle ear and defective hearing. The secondary infection of the ear can be explained by the orifices of the Eustachian tubes being on a level with the palate instead of just above this level. This lower location gives a more horizontal direction in the tubes as they pass outwards.

When the nasal passages are occluded by the adenoids, the bridge of the nose becomes swollen, and the nostrils small and narrow. If the obstruction be only partial the nostrils are frequently very large, because of their unconscious expansion in search of air. The palate is highly arched, and the irregular projecting teeth, and poor-developed under-jaw, are due to the constant mouth-breathing and the fact that the tongue does not exert lateral pressure on the palate as occurs in nasal breathing. There is chronic inflammation of the throat and a thick catarrhal wooden voice, lacking resonance. "Ninepence for fourpence" becomes "dinepens for forpens."

The digestive system is affected; the appetite may be poor or large. The child suffers from gastro-intestinal catarrh with sickness and vomiting. There may be constipation or there may be diarrhœa, with the passage of mucus, and, eventually, prolapse of the rectum. Other symptoms are cough, restless nights and incontinence of urine. The child becomes pale and peevish and has dark lines under the eyes. To a great extent I think that this condition is largely due to the swallowing of catarrhal mucus from adenoids, for adenoids secrete mucus quickly, which may pass into the stomach, especially during sleep. Food becomes mixed up with this mucus, and is either vomited or imperfectly digested. Imperfect absorption causes emaciation, rickets and anæmia. The child now becomes languid and dull. He is disinclined for exertion, and complains of weariness and depression, so that tuberculosis may be suspected.

There is an interrupted distribution of adenoid tissue throughout the whole length of the digestive tract, such as the irregular deposits of the stomach, the solitary lymphoid structures and agminated lymph-glands of the small intestine and the solitary glands of the large intestine. These structures may get out of order and set up that condition of subacute catarrh culminating in frequent bilious attacks, etc., met with in children between the ages of three and four years, and again between ten and twelve years. Where such conditions exist, definite evidence of adenoid overgrowth will also be found in the naso-pharynx. An improvement often synchronises with removal of the adenoids.

Adenoids cause other diseases and defects, possibly remote, but invariably associated with them. Notice at this stage the stoop shoulders and flat chest; the bleary, watery eyes from catarrh of the tear-ducts; the acute and frequent inflammations of the throat and increased liability to exanthems. Acute inflammation of the middle ear is produced by the poor circulation and blocked drainage of the middle ear and tubes. Frequent epistaxis and cervical adenitis occur because of the drainage into the cervical glands.

There is an increased liability to tuberculosis, with a train of nervous disorders from reflex irritations, such as headache, eye-strain, restlessness at night, disturbed sleep, bed-wetting, habit-spasm, twitchings of nose, lips and neck, shrugging o shoulders, and general nervous exhaustion, and even epilepsy. The chronic defective air-supply, poor nutrition and defective hearing complete the picture; and when the condition called aprosexia nasalis is reached the child is backward at school, and often sits in apparent apathy, oblivious to the movements of the children around.

Diagnosis.

Having regard to all the signs and symptoms present, it may be easy enough to presume that adenoids are present. To decide this seat the child on a chair or on the knees of an assistant facing a good light and alongside a good lamp. When the mouth is open, gently depress the tongue and look at the faucial tonsils. If they are large, if there are signs of lymphoid nodules on the walls of the pharynx, if on tilting the head backwards the movements of the soft palate are seen to be limited or the palate is fixed; then adenoids are probably present.

Posterior retinoscopy is not easy and seldom successful in child life, but palpation should be performed in all cases where there is presumptive evidence. Stand behind and to the right of the child, who is seated on a chair or on the knees of a nurse. Press the left cheek between the teeth with the thumb of the left hand. Then pass the tip of the right forefinger (protected if necessary by an india-rubber finger-guard) just behind the right posterior palatine arch, and then up to the top of the pharynx. (If the finger is inserted in the middle line the palate is firmly retracted against the posterior pharyngeal wall, so that the finger cannot be made to pass up into the posterior nasal space.) If adenoids are present a variable soft gelatinous and pliable mass is felt. It readily bleeds. As a rule adenoids are felt as a single mass in the middle line of the vault fornix or posterior wall.

Palpation with the finger must be done quickly, and no time given for the child to struggle and get excited. It is difficult when chronic inflammation has thickened the soft palate to such a degree that there is hardly any room between it and the posterior wall of the pharynx. The finger may find the roof free, while adenoids may lie undetected on the posterior wall of the nasal space. Again, no large mass may be felt, the adenoids being diffuse or overhanging Rosenmüller's forsæ, and thus escaping notice. Hypertrophied posterior turbinate bones may not only cause symptoms, but may be mistaken for adenoids. In palpating it is difficult to identify the anatomical features of the naso-pharynx, as such landmarks are far less defined in children and the subjects of adenoids.

Differential Diagnosis.

Recognise a polypus by its mobility, and see it in the nasal cavity. A fibroid tumour is round and hard and feels like a large split marble attached to the posterior wall of the pharynx. Malignant growths are rapid in formation and cause pain, fœtid discharge and wasting. The body of the axis often causes obstruction at the point of junction of nose and naso-pharynx by projecting into the oro-pharynx. Hypertrophied tonsils often cause symptoms simulating adenoids, in the dull thick speech, and by pressing back the posterior arches of the soft palate obstruct the end of Eustachian tubes and cause deafness. It is rare to find hypertrophied tonsils without hypertrophied lymphoid tissue in the naso-pharynx.

Structure.

Adenoids consist of a fibrous connective-tissue framework supporting masses of lymphoid cells, with an epithelial covering on its free surface. From this framework, fibrous septa pass through the substance of the gland, and ramify in all directions, forming a delicate reticulum enmeshed with numerous blood-vessels, and lymphatics, and clusters of mucous glands. Contraction of the fibrous tissue causes gradual shrinking of the adenoid mass. *This change is independent of the age of the patient.* It does not necessarily commence at or before puberty, but may occur at all ages, and may be more marked in the young child than in the adult. *There is no definite period for this shrinking to occur. To wait for it is foolish, for the secondary effects described above will progress.*

Prognosis.

If the affection is pronounced it usually becomes chronic. When children become deaf they may gradually lose the power of speech, and fall into a condition of hebetude which sometimes simulates idiocy; but they quickly improve when the cause is removed. If the condition is diagnosed and properly treated before irreparable injury has been done to the ear or the general development, the prognosis is good. The removal of adenoids often prevents serious ear complications, and improves the general health of the child.

Enlarged Tonsils.

In about one third of the cases of adenoids

there are also enlarged tonsils. Again, most cases of enlarged tonsils, as mentioned before, show adenoids. Tonsils may be abnormal because of excessive size, because of changes in their structure due to chronic inflammation, irregularities of their surface and acute and chronic infections. The tonsils have attracted considerable attention as channels of infection depending on their structure and surface regularity. When chronically inflamed tonsils may become a focus for the entrance of infective disease, such as diphtheria, rheumatism, etc. Primary tuberculosis of the tonsils has been noted in from 4 to 10 per cent. of tonsils removed.

Abnormal tonsils may be divided into:

(1) Hypertrophied tonsils of normal structure. The enlargement may be regarded as compensatory hypertrophy, to meet the need of lymphatic secretion.

(2) Hypertrophied tonsils the result of chronic inflammation. When the parenchyma or gland-tissue is specially affected, the soft more or less vascular tonsils, with crypts and much secretion, are the result.

(3) Chronically inflamed tonsils with an irregular cryptic surface. The tonsil may be hard and tough with few less prominent crypts, or very small with vascularity much diminished. This small diseased tonsil is more susceptible to infection than the younger or larger one, which has a better circulation. Food may become lodged in the crypts and undergo decomposition there. A tongue depressor carelessly applied will cause the act of gagging, and thus brings the tonsils towards the middle line, giving otherwise normal tonsils the appearance of being large and obstructive.

Enlarged tonsils may meet in the middle line yet produce few symptoms, provided the passage of air through the naso-pharynx is unobstructed. But even though the pharyngeal tonsil is not enlarged there will be difficulty in swallowing and thickness in speech. They will, however, cause obstruction in the naso-pharynx should they enlarge upwards and backwards. When this happens the same interference with growth and breathing takes place as when the obstruction is primarily in the naso-pharynx. In short, the symptoms are then similar to those of adenoids.

The consequences of enlarged tonsils are:

(*a*) Obstruction of breathing.

(*b*) Increased liability to throat infections. The pits or crypts are the incubators of germs. Exposure to cold, or indigestion or foul air provide the opportunity for the invasion of tonsillitis, diphtheria, scarlet fever and meningitis. Streptococci and staphylococci are almost constantly present in the crypts of the tonsils, which are veritable incubators for the absorption of septic matter.

(*c*) Enlargement of the glands of the neck.

(*d*) Indigestion, poor nutrition and lowered general health. Tonsillar crypts are often large, and the foul matter they contain is emptied into the throat and swallowed, causing indigestion. The crypts and fissures of the tonsil are, as has been stated, traps for microbes, and, because of the peculiar arrangement of the investing epithelium, open the gates to their invasion into the tissues of the tonsil, whence, through the lymphatic channels and vessels, they gain entrance into the system.

(*e*) Ear complications, extension of catarrh to the Eustachian tubes, or pressure and clogging of their orifices.

(*f*) Increased liability to tuberculosis. Strassman has reported thirteen cases of tuberculosis of the tonsils in twenty-one tuberculous cadavers. The infection may travel through lymph-glands of the neck, then into the deeper glands, and then on to the pleura and apex of the lung.

(*g*) Increased liability to heart disease. Acute tonsillitis is a general infection in which poisons have passed into the system. Tonsillitis and rheumatism exist together in some intimate relation. Clinical observation supports the view that rheumatic fever originates in acute tonsillitis.

(*h*) Acute suppurative, osteo-myelitis, septic thrombo-phlebitis, pulmonary gangrene and other infectious conditions have their initial lesions in tonsils, and may be due to an infection by the same route.

Treatment.

In the 'British Medical Journal' of May 13th Dr. Steuart advises the reduction of enlarged tonsils and adenoids by means of X rays. I have not seen any successful case treated in this way. No doubt after several sittings a form of atrophy takes place. Dr. Ashby, of Manchester, advises the administration of lymphatic gland extract in 5-gr. doses thrice a day. In compensatory hypertrophy this form of treatment is worthy of trial.

I have several cases under observation in which children, of ages ranging between four and six years, are taking 5 gr. of lymphatic gland extract.

I cannot see how an enlarged pharyngeal or faucial tonsil which has undergone a pathological hypertrophy, can have any physiological function whatever. To my mind they are simply foreign bodies. There is but one method of treatment worthy of the name—removal of the growths.

It is customary to consider the operation for the removal of adenoids and tonsils as trivial; but it demands personal dexterity and experience, and requires also the intelligent co-operation of both operator and assistants. Without the help of an expert anæsthetist the operation should not be attempted. In hospital practice, where many cases have to be operated upon quickly, it is the practice to subject the patients to short anæsthesia. Ethyl chloride is the favourite anæsthetic. Skilfully administered, this is perfectly safe. I have not been able to find the record of one fatal case in the hospital, when this vapour is given, in from 700 to 800 cases yearly. An excellent paper on ethyl chloride anæsthesia, by the senior anæsthetist, Dr. Hadfield, appeared in the CLINICAL JOURNAL of April 2nd, and should be carefully read. Granted, then, the sympathetic help of the anæsthetist, and scrupulous attention to details, the operation is a success.

It is unnecessary to discuss all the different measures and instruments which have been employed for the removal of adenoids and tonsils. The guillotines alone invented or modified are legion. I have had one made by Messrs. Meyer & Meltzer, modified from Ballenger and built on the scissors principle, but success depends not so much upon the instruments used, as upon the intelligent and sympathetic and quick co-operation of the operator, the anæsthetist, and the nurse; and, finally, upon a thorough familiarity of the parts to be operated upon.

In this hospital the operation is performed upon a table before a good light, and the patients have been previously prepared in the usual way. Attention is paid as far as possible to the previous cleansing of the nose, mouth and ears; and they are in as good condition as possible. The instruments used are:

(1) Mouth-gag for the lateral teeth.

(2) Curettes, caged and plain, in graduated sizes.

(3) Post-nasal forceps.

(4) Heath's guillotines in graduated sizes, with thin rings, and the blades cutting close up to the outer or upper aspect of the instruments. The shafts are strong and unbendable.

(5) Iced cold water with sponges and swabs. A solution of adrenalin, 1 in 10,000, together with a tracheotomy set, and other necessaries are also at hand.

I endeavour to enucleate the tonsils according to the method of Greenfield Sluder, of St. Louis. The outer aspect of the guillotine is applied to the tonsil and threaded round it as you push the gland on to the alveolar margin of the jaw, so that the gland can be pushed through the ring of the instrument. The method may readily be remembered by the mnemonic: O utwards, U pwards, F orwards, being the directions in which you move the guillotine after threading it round the tonsil, and during which process the instrument quickly travels around a half-circle, so that ultimately the outer or upper aspect looks towards the roof of the mouth, the handle meantime having been carried to the opposite angle of the mouth and slightly depressed.

The light should be good. The patient lies with his back on the table with the face partially turned to the right. This is the position adopted at the Prince of Wales's Hospital. First insert the gag, and extend it when the apparatus for anæsthesia has been removed. The mouth should be opened wide enough to give a good view and easy admittance to the tonsils. The right hand holds the handle of the instrument firmly, and the right tonsil is removed first. Now, having grasped the handle of the guillotine firmly, introduce it edgewise, using it as a tongue-depressor, and with the outer aspect of the ring looking towards the right tonsil. Encircle the lower pole of the tonsil, and press the instrument firmly outwards, at the same time raising the tonsil forwards as far as you can, helping this movement by using the second joint of right forefinger as a lever to the shaft. During this manœuvre you are quickly and deftly turning the shaft of the instrument through nearly half a circle to the right, and carrying the handle to the opposite angle of the mouth. The outer aspect of the shaft now looks upwards to the roof of the mouth. While depressing the handle slightly, quickly push the ring outwards and upwards behind the tonsil, and the position of affairs will be:

(1) The anterior pillar of the fauces is bulging forwards.

(2) The ring of the instrument is upwards and behind the tonsil.

(3) The tonsil is resting on the ring of the instrument.

Now withdraw the instrument slightly. At the same time press firmly on the anterior faucial pillar with the thumb of left hand, forcing the tonsil through the ring of the guillotine. Should the tonsil be moderately big, a sound like the reduction of a dislocated joint is heard as the gland is being forced through the ring. Push the blade of the guillotine home, and it will enter between the anterior pillar of the fauces and the capsule of the tonsil. Now turn the shaft and handle of the instrument outwards to the left, and withdraw the tonsil, plus its capsule, resting on the inner or under aspect of the guillotine.

In the same way approach the left tonsil before the view is obscured by blood. Now turn the patient over on his face and free the mouth and throat of blood. Quickly turn him on his right side, the anæsthetist fixing the head and slightly flexing it.

The operator now passes his left forefinger behind the soft palate to protect it and draw it forward. With the right hand push the caged curette back to the pharynx. Carry up in the middle line till it touches the posterior border of the septum, the curette resting like a pen in the right hand with a strong forefinger on the upper surface of the instrument, and with a firm, easy and gradual pressure push it backwards, downwards and outwards with a sweeping movement. The main mass of adenoids is always withdrawn in the cage of the instrument. Any remaining fragments of adenoids are now removed with the short, quick scrapes of an ordinary curette. Introduce now the right forefinger, the nail of which should be of carefully prepared length and dipped in absolute alcohol. Explore the choanæ. Push the finger in to be sure of their patency. If not quite free, scrape with the finger-nail and clear. Quickly remove the gag and allow the blood to escape freely through nose and mouth. Revive the patient by means of iced water.

Hospital patients are kept quiet for three hours, and if well, and no hemorrhage, they are allowed to go home. They are asked to rest in bed for two days and kept at home from school for seven days, and dieted on semi-solid foods.

Afterwards, as far as possible, the patient is asked to exercise twice daily for fifteen minutes by gently swinging arms around and taking deep long breaths through the nose. This is continued for at least three months.

With proper care I have not seen many complications follow the operation. It is not, as it is commonly supposed to be, devoid of danger. The most frequent and dangerous of these complications may be divided into two groups: (1) hæmorrhage; (2) infections. In 1907 Burrell and Orr reported seven fatal cases of hæmorrhage. Six cases of septicæmia following the operation have been recorded, and three cases of endocarditis. If patients are carefully watched, however, such sequelæ may be reduced to a minimum.

Patients receive the greatest relief both immediately and in the hereafter by the removal of the growths in suitable cases. It is gratifying to see the miserable, white-faced, open-mouthed, deaf catarrhal child, blossom forth and become almost robust in a comparatively short time after the operation; the change in the facial aspect being quite remarkable. An incalculable number of individuals at this present moment are useful members of society, who would otherwise have become permanently deaf, besides suffering great inconvenience and discomfort. On the other hand, an enormous number of deaf people owe their affliction to neglect of this operation in early childhood.

August 25th, 1913.

RADIUM AS EMPLOYED IN THE TREATMENT OF CANCER, ANGIOMATA, KELOID, LOCAL TUBERCULOSIS AND OTHER AFFECTIONS. By LOUIS WICKHAM and PAUL DEGRAIS. Translated by A. and A. G. BATEMAN, M.B., C.M. London: Adlard & Son, 1913. Pp. 111. Price 2*s.* 6*d.* net.

M. Louis Wickham, the pioneer of radium therapy, in 1906 founded the "Laboratoire Biologique du Radium de Paris," where most of the radium therapists of the world have been instructed. Here also M. Degrais has worked for many years as a colleague. Their object is not to describe in detail the technique of radium therapy, but to show some of its results. The origin and method of preparation and the physical properties of radium are briefly but clearly explained. The radium emanation, the phenomenon of induced radio-activity, the different varieties of rays (α, β, γ) and their passage through substances are then considered. The methods of therapeutic filtration and "cross-fire" are described. A very brief account of appliances is given. The reactions produced in tissues by the radium are then discussed. They manifest themselves clinically in two ways—in (*a*) the inflammatory reaction, and (*b*) the selective and non-inflammatory reaction. It is on the latter that the therapeutic value of radium depends, for diseased tissues are often peculiarly sensitive to the rays. In cancer-cells cytolysis is produced, and in favourable cases they are replaced by connective tissue. A large number of illustrations is given, showing the curative results obtained in cancer, sarcoma, angioma, keloid, tuberculosis, including lupus. We note that in cancers of the buccal cavity "many palliative results," but also "many failures" have been obtained. By permission of the publishers we give a number of illustrations, showing some of the most remarkable results.

The book should prove interesting to the pro-

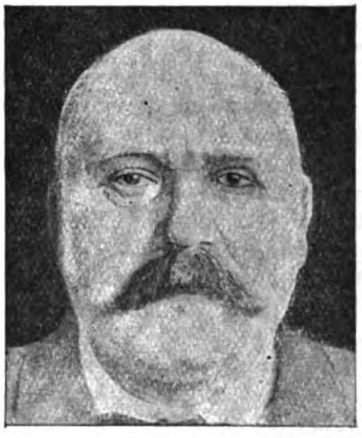
Enormous erectile nævus before treatment.

After treatment.

Lupus vulgaris before treatment.

After treatment.

Epithelioma before treatment.



After treatment.

fession generally. It is clearly and concisely written, and well illustrated. The translation has been excellently done. If radium therapy has not justified all the high hopes that were entertained, it has proved a powerful addition to our therapeutic armamentarium.

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A LECTURE

ON

PNEUMOTHORAX.*

By J. WALTER CARR, M.D., F.R.C.P.,

Physician to the Royal Free Hospital and to the Victoria Hospital for Children, Chelsea.

Ladies and Gentlemen,—I propose to deal with pneumothorax mainly from the point of view of diagnosis; and what a simple matter, merely from the theoretical standpoint, this seems to be. The mental picture we form of a case of pneumothorax is probably somewhat as follows: We think of a patient who is taken suddenly ill with a sharp pain in the side and very severe dyspnœa, and who passes into a condition of collapse which may prove fatal. If he survives, so that we are able to make a thorough examination, we expect to find the heart markedly displaced; and one side of the chest fixed and over-distended, giving a very hyper-resonant percussion note and no breath-sounds. The history, the symptoms and the physical signs are all so perfectly characteristic that it seems hardly possible to make a mistake. Yet, as it happens, most of the cases of pneumothorax which I have been asked to see during the last five or six years have escaped recognition in their earlier stages. Why is this? The condition, according to our text-books, appears absolutely typical; yet experience shows that errors are very frequently made. Sometimes the reason is obvious. For instance, there is the localised pneumothorax: the lung is fixed by adhesions, so that when perforation occurs into the pleural cavity it cannot collapse altogether, and the patient gets a localised cavity in the pleura, which, very naturally, may be hardly distinguishable from a cavity in the lung itself. Again, we meet, not uncommonly, with a case of this sort: A patient is

* Delivered at the Polyclinic.

suffering from advanced phthisis and obviously going downhill. After a time the downward progress undergoes a definite acceleration; there is no very marked change in the symptoms, except perhaps more rapid breathing, but the man's general condition quickly deteriorates, and he is so weak that no thorough examination is made. Presently death occurs, and on the post-mortem table we find to our surprise a pneumothorax. On looking back over the history of the case we can very likely recognise the time at which it arose; but the patient was already so ill that it did not give rise to any sudden or marked alteration in his condition, and consequently its existence was not suspected. Even if such a case is correctly diagnosed the result must be the same, for nothing can be done to prolong the patient's life. These cases may be compared with those of perforation of the intestine in a late stage of severe typhoid fever: cases in which the patient is already so desperately ill that it is difficult or impossible to recognise any additional signs or symptoms produced by the perforation, and even if they are recognised an operation must necessarily be futile. Furthermore, there are some cases of pneumothorax—I shall refer to one specially later on—in which the patient is so ill when we see him that a careful physical examination is practically impossible. We have to do the best we can, having regard to his serious condition, but that often means that we must necessarily be unable to make an accurate diagnosis.

Unfortunately, however, pneumothorax is often overlooked when none of the difficulties just referred to is present; when the condition is not a localised one, when it does not supervene in a patient already very ill, and when there are no special reasons for not making a thorough physical examination. Why are mistakes made under such circumstances? First of all, I think, because pneumothorax is a comparatively rare condition. We may see very many patients without meeting with it. Consequently we are not on the look-out for it; and unless it almost obtrudes itself upon us, it is very easy not even to think of the possibility of pneumothorax; and if we do not think of its possibility we are likely to overlook it.

Secondly, we commonly associate the onset of pneumothorax with very definite and severe symptoms; I have referred to them already—severe pain in the side, urgent dyspnœa, and more or less collapse. As a matter of fact, however, pneumothorax, even when complete, may cause surprisingly slight symptoms, and is then sometimes spoken of as "latent pneumothorax." Some time ago I saw a man, æt. 22 years, who four days before, when getting up in the morning, had been seized with a sudden pain in the left side and some dyspnœa on exertion, not very severe, and not enough to incapacitate him from doing his work as a clerk, although probably he could not have undertaken severe manual labour. He walked to my house, and when he entered my room looked fairly comfortable; but on examination I found he had a complete left pneumothorax, which had obviously developed four days before when the pain first started. Let me refer to another case. A man, æt. 52 years, was sent to me by a very able practitioner because he had a pleural effusion which was clearing up, but his condition was not satisfactory and the physical signs in his chest were somewhat abnormal. He said that about a month before I saw him he had had some pain in his right side; in a few days it got better, then became rather worse again and he called in his medical man, who found a pleural effusion and treated him accordingly. When I saw him he still had a small effusion, which cleared up a few weeks later, but he had also a complete right pneumothorax. Exactly when that pneumothorax developed no one could say.

A third reason why the condition is often overlooked—and probably the most important of all—is the extraordinary variability in the physical signs. Let us take the more important signs one by one, in the order in which we expect to find them when making a routine physical examination of the chest. First of all, we say the affected side should be over-distended—more distended than it can be in health. Quite true, as a general rule, but it may not be distended at all; it may, indeed, be somewhat contracted, at any rate in those cases in which there is a perfectly free opening from the lung into the pleural cavity, so that there can be no positive intra-pleural tension. Secondly, we say that on the affected side there should be complete loss of the respiratory movements. Quite true. Yet in a certain number of the cases the movements are perfectly good, viz. in those patients who have a free opening between

the lung and the pleural cavity, so that with each inspiration the air passes directly into the pleural cavity and out again with each expiration, the lung not being distended at all; the pleural cavity merely fills and empties with each inspiration and expiration respectively. Thirdly, we expect to find the heart displaced, often markedly so. Quite true; nearly always it is, especially when the pneumothorax is on the left side. But it is sometimes very difficult to be quite sure about the displacement, for, as a result of the shock caused by the pneumothorax, the heart's impulse may be very feeble or even imperceptible, whilst the unaffected lung may become somewhat over-distended and overlap the heart, so that there may be very little cardiac dulness. The result is that even though the heart may be much displaced, yet it may be difficult, or even impossible by physical examination only, to be quite certain about it.

Fourthly, we expect to obtain a tympanitic percussion note. Quite true; usually we do. Some good authorities state, however, that if the tension in the pleural cavity gets very high indeed, the excessive distension may lead to a muffling of the note, so that it becomes toneless and almost dull. I believe that such a condition is very rare; I have not met with an example myself. We know, however, that as a result of pneumothorax a pleural effusion very frequently follows, and this, of course, gives a dull note, and sometimes we are content with finding the effusion, and go no further in the diagnosis; we fail to recognise that the effusion is secondary to a pneumothorax. We may have learned, moreover, from experience, that in the presence of an ordinary pleural effusion the physical signs over the lung above it are often very misleading, so that even though we may suspect the presence of a pneumothorax also, we feel unable to arrive at a certain diagnosis. To this question of the difficulties which arise in connection with the diagnosis of a pneumothorax associated with pleural effusion I shall refer later.

Fifthly, we come to auscultation. We expect breath-sounds and vocal resonance to be absent over a pneumothorax. Quite true; yet in a minority of the cases the breathing is markedly bronchial or even amphoric, and the vocal resonance much increased. Thus in one case of pneumothorax we may get complete loss of breath-sounds and of vocal resonance, whilst in another we may hear loud amphoric breathing and bronchophony. Why this should be is difficult to say, and I will not attempt an explanation; but remember we may meet with either of the two extremes, or possibly with very weak and distant bronchial or amphoric breathing.

Lastly, over a pneumothorax we expect to hear three special auscultatory signs. First, there is what is known as the "bell-sound" or "coin-sound," produced by tapping two coins placed on the surface of the chest, whilst listening over another part of the pleural cavity; if a pneumothorax is present, we hear the characteristic clear ringing sound. The second special sign is the "succussion splash," only present, of course, when there is an effusion as well as a pneumothorax. We shake the patient gently and hear the fluid splashing in his pleural cavity. Thirdly, a less important sign is known as the "metallic tinkle," a peculiar echoing sound like that produced by a drop of water falling in a cave or grotto. This is not so important as the other two. When these signs are present they are fairly characteristic, though not absolutely pathognomonic of pneumothorax. The coin-sound may be heard over a large cavity, and the succussion splash may be obtained shortly after a patient with a dilated stomach has drunk some water; so that we must beware of mistaking a dilated stomach for a pneumothorax merely because a succussion splash can be obtained. Note also that the succussion splash is less easily obtained with a purulent than it is with a serous effusion, as pus is not so easily thrown into vibrations. Occasionally, however, we meet with a pneumothorax in which every one of these special signs is absent.

We have discussed some of the chief difficulties which may be met with in connection with the diagnosis of air in the pleural cavity. Let us now consider further some of the problems which arise when, as so often happens, an effusion develops secondarily to the pneumothorax.

Such an effusion may be helpful in the diagnosis or it may be misleading. First, it is helpful because when associated with a pneumothorax it moves freely in the pleural cavity with any change in the patient's posture, just as ascitic fluid moves freely in the abdomen, so that the fluid and the consequent dulness are always in the most de-

pendent part of the chest or abdomen, as the case may be. In this respect there is a marked contrast to the behaviour of an ordinary pleural effusion, for the latter shifts very little with any change of posture of the patient—so little that we can almost ignore any change in the distribution of dulness which results; but it is obvious that when the pleural cavity contains air any fluid present is absolutely free to move in any direction in which it is influenced by gravity.

Secondly, note that we may have a considerable effusion along with air in the pleural cavity and yet very little dulness. Contrast the condition of affairs in the ordinary pleural effusion with that when a pneumothorax is present also. In the former the diaphragm remains very much in its normal position, with its convexity upwards, and the effusion, as it accumulates, fills up the angle between the arch of the diaphragm and the chest-wall, so that a comparatively small quantity will give a relatively considerable area of dulness. Now consider the condition in a case of complete pneumothorax. The diaphragm on the affected side, instead of being arched upwards, is depressed or cup-shaped. The effusion fills up this cup, but a considerable quantity may accumulate in it before sufficient comes into contact with the chest-wall to produce any extensive area of dulness.

A third point about a pleural effusion secondary to pneumothorax is that, apart from the difficulty in connection with the cup-shaped depression of the diaphragm, the dulness produced by a moderate quantity of fluid may be easily missed altogether, owing to its free mobility. Suppose we have to deal with a patient—and it is often the case in pneumothorax—who is so ill that he cannot be sat up in bed for physical examination, but has to be kept horizontal. We examine the front of the chest while the patient is lying on his back, and of course get no sign of the presence of fluid there, because it is all lying posteriorly; we find merely the ordinary signs of air in the pleural cavity. Now, turning the patient over on to his face, we examine the back, and as the fluid naturally goes over at the same time, the back becomes resonant, owing to the fluid having gravitated entirely to the front of the chest. Consequently, whichever way we turn the patient, the fluid evades us. Thus a moderate-sized effusion may be present without being discovered if the patient is examined only whilst in the recumbent posture. Of course, if he can be sat up the difficulty is very much less; but it not uncommonly happens that he is too ill for this to be done with safety. This is a point which you will rarely find mentioned in books, but I know from experience that it is of practical importance.

Now, to return to the question of the abnormal signs which are often present over the lung above an ordinary pleural effusion, and to the difficulties they may give rise to in connection with the diagnosis of pneumothorax. We know that in the case of a patient suffering from ordinary pleurisy with effusion the physical signs at the apex of the lung on the affected side are most unreliable. There may be a peculiar hyper-resonant note known as the skodaic note, very much like the tympanitic note over a pneumothorax. The auscultatory signs also may be greatly modified. We may hear bronchial, or even amphoric, breathing, which may suggest, on the one hand, a tuberculous lesion at the apex, or, on the other hand, the possibility of pneumothorax. More than once I have been led, in connection with a pleural effusion, into a pretty confident diagnosis of tuberculous mischief at the apex of the lung; but as the fluid has been gradually absorbed these abnormal signs have entirely disappeared. On the other hand, it is easy to imagine that the unusual signs—the skodaic resonance and the altered breath-sounds—may be due to a pneumothorax. One of the most helpful points in the diagnosis of such cases is that with an ordinary pleural effusion the fluid does not move when the patient alters his position; whereas if a pneumothorax is present, of course it does. Hence the importance to be attached to shifting dulness in the diagnosis of a pneumothorax.

Finally, recognising these difficultes, what are the points to which we should attach the greatest importance in making a diagnosis? First, displacement of the heart. I have already mentioned how difficult it is sometimes to be sure of the displacement; but we should never be satisfied until, in any doubtful case, we have determined, as accurately as possible, the position of the heart in the thorax. Marked downward displacement of the liver or spleen may also be an important sign. Second, a hyper-resonant percussion note over one side of the chest. There is no excuse for making a mistake in the diagnosis between

pneumothorax and emphysema. It is true there is a hyper-resonant note in both; but in emphysema it is equal on the two sides. It may be impossible by physical examination in a case of emphysema to ascertain the position of the heart, but that does not matter as long as the percussion note is the same on the two sides. Third, the presence of shifting dulness—a sign that is, of course, only available when there is a secondary effusion, and we must remember that pneumothorax may occur without being followed by any effusion. Fourth, breath-sounds which are either absent or, less commonly, amphoric; these are the two extremes; but, at any rate, the breath-sounds are obviously abnormal and markedly different from those heard over the healthy lung. We must bear in mind that the breath-sounds on the unaffected side are usually harsh or compensatory, and this makes the difference between the sounds on the two sides all the more marked, especially when they are weak or absent over the pneumothorax. Fifth, the presence of any one of the special auscultatory signs already described—the coin or bell-sound, the succussion splash and the metallic tinkle. It is probably exceptional for all three of these signs to be absent, although it is also rare for the three to be present at the same time. In a doubtful case, radiography may settle the diagnosis, often showing up very clearly indeed the pleural cavity filled with air, the displaced heart, the collapsed lung, and if an effusion is present, exhibiting very distinctly the contrast between it and the air above, and also the vibrations in the fluid when the patient moves. Thus, by looking at the patient's chest with the fluorescent screen, a diagnosis may often be made at once.

There will always be some cases of extreme difficulty. I have already referred to the trouble often met with in distinguishing between a large cavity in the lung and a localised pneumothorax. Obviously the signs over the two may be well-nigh identical. With a cavity, however, there is not likely to be any displacement of organs, and the note is hardly ever definitely hyper-resonant. Sometimes difficulty occurs in connection with traumatic rupture of the diaphragm. I remember, some years ago, being asked by a surgical colleague to see with him a young man who had been admitted to hospital the previous day, having been nipped between the buffers of two railway carriages. On examining the patient's chest I found that on the left side there were distension, loss of movement, a hyper-resonant percussion note, and absent breath-sounds, and that the heart was displaced. Almost without hesitation I said he had got a pneumothorax—a condition which one might well expect as a result of such an accident. A few days later the man died. At the post-mortem it was found that his diaphragm was ruptured on the left side and that his stomach nearly filled the left pleural cavity. There was no pneumothorax. I hardly know how an accurate diagnosis could have been made in such a case. Perhaps if a very thorough physical examination had been made, one might have come across some discrepancy which would have suggested that the condition was not a pneumothorax, but as a rule in these cases the patient's condition is such as to render a complete examination impossible. On the other hand, the recognition of such a condition is of some importance from the point of view of operative interference. Had one suspected that the man's stomach was in his pleural cavity, his abdomen might have been opened and the displaced organ replaced in its normal position. The risks, under the circumstances, would have been very great, and probably the man would not have survived the operation. On the other hand, it is obvious that if in such a case nothing is done the patient is bound to die; consequently we cannot make matters any worse by an exploration. That case has always been a forcible reminder to me of the great difficulties which may occur in connection with accidents which are almost equally likely to cause either pneumothorax or else rupture of the diaphragm with such consequences as those above described.

As another example of difficult or impossible diagnosis I may describe a very interesting case which I saw about two years ago. The patient was a lady, æt. 41 years, who for five years had complained of dyspnœa on exertion. Nothing had ever been found wrong with her lungs, but she was supposed to have a feeble heart. On June 26th, 1911, being then about two months pregnant, she began to complain of rapidly increasing dyspnœa. Nothing abnormal was found in her lungs. She quickly got worse and I saw her on June 30th. She was lying on her right side, and was apparently only kept alive by the continuous administration of

oxygen; the slightest attempt to place her in any other position produced the utmost distress. Under the circumstances it was quite impossible to make any adequate physical examination. I could not determine whether her heart was displaced or not. Its action was very rapid, but the sounds were normal and no murmur could be detected. The only definite abnormal sign I could make out was deficient air-entry into the left lung. The question of pneumothorax was discussed, but on the whole the conditions present did not seem to justify such a diagnosis, and I am inclined to the view that it was a case of cardiac or pulmonary thrombosis. For three days she remained in this condition, lying always on her right side, being given oxygen almost without intermission, frequent injections of strychnine, and occasional small injections of morphine. Then she began slowly to improve. Abortion followed, and when the chest was further examined it was found that vocal fremitus, vocal resonance and the breath-sounds were all distinctly weaker than normal on the left side. On the right side the breath-sounds were somewhat exaggerated, but the position of the heart could still not be accurately determined; there was no pleural effusion. Similar attacks, somewhat slighter in character, recurred in the following August and in November. A fourth in April, 1912, proved fatal. Between the attacks the patient rallied sufficiently to walk from one room to another. With each attack there was deficient air-entry into the left lung, but the breath-sounds became fully re-established between the attacks. Sometimes the heart was found displaced to the right; there was no hyper-resonance and no bell-sound. After the first attack the patient was seen several times by Sir Douglas Powell, who was able to make a far more complete examination than I could. He noted that the breath-sounds were markedly feeble over the left lung, but with no bronchial quality, and thought there was some œdema at the base of that lung and some enlargement of the right side of the heart. He never made more than a tentative diagnosis, but, on the whole, was disposed to think that the attacks of dyspnœa were caused by multiple, small pulmonary emboli, a result either of some septic condition of the uterus or of clotting in the right auricle. Points against this diagnosis were the absence of pyrexia and of blood-stained sputum. For two months before death bronchial breathing and crackling crepitations were heard at the left apex. At the post-mortem examination a left pneumothorax was found. The apex of the left lung was adherent; the lung itself only occupied about a quarter of its normal space, and there were scattered tubercles in both lungs, especially the left. The diaphragm was depressed on the left side; there was no pleural effusion. Even after the post-mortem exactly what had happened remains somewhat doubtful, but probably with each attack of severe dyspnœa a left pneumothorax had developed, whilst between the attacks the lung had re-expanded satisfactorily. The pneumothorax was doubtless due to the tuberculous disease of the lung; but although this was slowly progressing, it never caused any fever and only slight physical signs towards the end of life. In such a case probably an accurate diagnosis was impossible, and it serves very well to illustrate the great difficulties which we may have to encounter. The fact that the patient breathed best when lying on her right (or healthy) side was a curious feature.

When we have recognised the presence of a pneumothorax the diagnosis is still incomplete, for we have still to consider what is the cause of the condition. In the vast majority of instances where the cause is not an obvious one, such as trauma or malignant disease, pneumothorax is due to tuberculous disease of the lung. We hear sometimes of the possibility of an emphysematous bulla on the surface of the lung rupturing and causing pneumothorax. No doubt when we see such bullæ on the post-mortem table we are apt to think how easily they may rupture. As a matter of fact, however thin they may be, they probably very rarely give way, because during life they are supported by the chest-wall. Again, we not uncommonly hear of pneumothorax resulting in a previously healthy young man from a severe muscular strain. Certainly it is not impossible for such a strain to give rise to pneumothorax, but it very rarely does, and it is much more likely to cause subcutaneous emphysema. In the vast majority of instances, when we hear of pneumothorax produced by a muscular strain in a man apparently healthy, it means he had some latent tubercle, and that probably a small caseous focus has ruptured through the pleura and so given rise to the pneumothorax. The important point to emphasise, therefore, is that every unexplained case of

pneumothorax should be regarded and treated as being probably tuberculous in origin. Very occasionally we meet with pneumothorax in young children, and then probably the most frequent cause is a septic broncho-pneumonia. I saw one such case a few years ago in a child two years old, with obvious broncho-pneumonia. After a few days' illness signs developed at the right base suggestive either of a large cavity or of a pneumothorax, and it soon became evident that the latter condition was present. Especially noticeable was the marked displacement of the liver, which was pushed down almost entirely below the ribs. I may say, in passing, that such marked displacement of the liver or spleen hardly ever occurs as the result of an ordinary simple pleural effusion. At the autopsy there was found to be very extensive broncho-pneumonia, and on the right side a small patch had softened and ruptured into the pleural cavity, and so established a communication with the bronchial tubes. Pneumothorax, however, very rarely arises in this fashion in adults.

Lastly, a few words as to one or two points in treatment. I need not discuss the treatment of the severe pain, the dyspnœa and the collapse which are often met with at the outset and not uncommonly prove fatal. When urgent symptoms have subsided, but the patient presents well-marked signs of pneumothorax, what shall we do? As long as no effusion occurs, let things alone. To what extent the lung will re-expand is always doubtful. Sometimes, especially in previously healthy patients, almost complete re-expansion occurs; in other instances it is only partial, but none the less, the symptoms gradually improve, and the patient, although incapacitated by shortness of breath from severe exertion, is able to lead a comfortable and useful life. What are we to do if effusion occurs? If clear fluid forms, as ascertained by puncture, we may leave it alone, for it is often re-absorbed in a few days, or at most a few weeks, although probably not so rapidly as an ordinary pleural effusion. If it is not absorbed, we can draw it off by simple syphonage. Even if it is semi-purulent syphonage will often suffice, and after two or three withdrawals it may cease to re-accumulate. I may mention the case of a man, æt. 22 years, who was admitted to the Royal Free Hospital in May, 1912. He had symptoms which suggested phthisis of several months' standing. At his right apex were well-marked signs of tuberculous disease, and on the left side complete pneumothorax with a good deal of free fluid. The effusion rapidly increased, and a week after admission 60 oz. were withdrawn. It was opalescent, and contained a large number of pus-cells. On June 18th 30 oz. more were withdrawn, and on July 11th a similar quantity; on this third occasion the fluid was almost clear. The man's temperature, which had been very irregular during the first fortnight following admission, had become and remained normal and his general condition rapidly improved. On July 25th he was discharged. During the month prior to his discharge he gained 17 lb. in weight. The signs of tuberculous disease in the right apex had almost disappeared; the left side still contained some fluid and the lung had only partially re-expanded. His general condition was exceedingly good, and he could get about actively with very little discomfort. If, however, the fluid is pure pus, and especially if it is associated with fever, then, if after one or two removals by syphonage it re-accumulates and the general condition of the patient remains unsatisfactory, it may be better to open and drain freely, just as one would in the case of an ordinary empyema. Fœtid pus should be drained freely without delay.

Every case, however, presents its own difficulties, and must be dealt with on its merits, so that it is better not to lay down too hard-and-fast rules.

September 1st, 1913.

Traumatic Denudation of the Male Genital Organs.—The front of the trousers and the skin over protruding parts were caught in a thrashing machine and torn off. The wound was so clean that a plastic operation could be done at once. Belfrage pushed the testicles up into a small pocket made for them in the lower part of the abdominal wall. The penis was covered with Thiersch flaps. The patient has never felt the slightest disturbance from the transplanted testicles. He already had three children and is well satisfied with the result. —*Journ. A.M.A.*, vol. lxi, p. 378.

A CASE OF HEAD INJURY.*

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GENTLEMEN,—The patient whom you have just seen submitted to immediate operation was admitted an hour ago, when he gave the following coherent account of his accident. Crossing the road he was knocked down by a motor-bicycle, being thrown violently down on his right side. He believes that his head struck the edge of the kerb and for the moment he was in a dazed condition, although not rendered unconscious. He was placed in a cab and conveyed to the house of a medical man, who dressed his face, which was very severely contused, the right eye being closed and blackened. Blood was at this time dripping from his nose, but there was no scalp wound. He was then sent on to the hospital and was seen by the house-surgeon, who found an effusion of blood over the right parietal bone extending down over the cheek and neck. There was crepitus over the malar bone and a small laceration of the tragus. No blood was found in the mouth, nor was there any evidence of blood coming from the deep part of the auditory meatus.

The patient was put to bed perfectly conscious and his pulse was slightly above normal and rather full; there were at this time no pupillary signs, nor had he been sick. Half an hour later I was called to see him and I then found him comatose; his breathing was slightly stertorous and about 25; his pulse was full, forcible and about 55; both arms and legs were completely spastic, the former being extended downwards and crossed so firmly in front of his abdomen that they could not be moved apart. All his muscle and tendon reflexes were entirely abolished, and from time to time there were slight convulsive movements of the thumb and forefinger of the left hand. On examining his eyes the corneal reflex in both was abolished and the right pupil was fully dilated, the left being contracted nearly to pin-point; there was no nystagmus.

* A Lecture delivered in the London Post-graduate School of Clinical Medicine.

On firmly palpating the cranial vault I found a distinct depression over the anterior inferior angle of the right parietal bone, which was almost obscured by the presence of a large cephalhæmatoma. There was effusion of blood into the right orbit, a subconjunctival hæmatoma obscuring the sclerotic over the outer two thirds of its area.

The signs I have detailed to you pointed quite clearly to a grave intra-cranial lesion, and the questions arising in one's mind were (1) was the condition due solely to right-sided meningeal hæmorrhage, or to a basal fracture and hæmorrhage as well? (2) If due to the first only, was the case too far advanced to admit of recovery after operation? (3) If the second contingency was suspected, what was the direction and extent of the fracture? That the injury was of the right side admitted of little doubt in this case, in view of the extensive contusion of the scalp and comminution of the malar bone of that side. The marked spasm of both sides of the body might in other circumstances have left one in doubt were it not for the convulsive twitching now and then occurring on the left side, and the very marked pupillary evidence in favour of right-sided hæmorrhage. The question of fracture of the base was not greatly in doubt so far as the anterior fossa was concerned, the orbital hæmorrhage being strongly indicative of it. The universal spasm of the limbs and the very rapid supervention of coma suggested something more extensive and rapid, but there was no other sign to support the belief, for there was no hæmorrhage from the ears or mouth, nor any evidence of intracranial nerve lesion. The diagnosis was therefore that of rupture of the great meningeal artery, with probably an extensive and rapid extra-dural hæmorrhage, since the coma was sudden and the paralysis rapid and extensive.

The patient was immediately removed to the operating theatre, and his hair being rapidly clipped off over the right side a large flap was turned down, the incision running from behind the pinna to the outer third of the supra-orbital ridge and rising nearly to the vertex. This flap included everything down to the skull. A 1-in. trephine was applied over the depression and the circle of bone removed. Immediately a mass of clot bulged into the orifice, which was enlarged with a pair of Hofmann's forceps to the size of a five-shilling

piece. Through this aperture a large mass of clot was removed by means of a scoop. The dura mater was flaccid and depressed about 3 cm. below the level of the skull and showed no immediate sign of bulging. There was a forcible double jet of arterial blood at the anterior end of the cavity from the two severed branches of the great meningeal artery. This point of severance lay so low down that the incision had to be carried down almost to the fractured malar and more of the great wing of the sphenoid had to be removed. The vessels were then caught by two pairs of artery forceps, which were left *in sitû*. The wound was lightly filled with gauze, and the scalp being replaced was sutured loosely by four salmon-gut sutures and the patient returned to bed.

From the moment that the clot was removed the spasm of the muscles disappeared; the pupils at first remained the same, but by the end of the operation the right pupil had begun to contract slightly, and there was a laryngeal reflex and some voluntary movements of the head and arms.

The subsequent history of this case is as follows. Within twelve hours the brain had expanded to its normal position, the pupils had become equal and of medium size; there was neither paresis nor spasticity, but he remained completely unconscious, his temperature varying from 99° to 100·5 F. At the end of forty-eight hours his wound was dressed, the gauze and forceps being removed. There was no bleeding and accordingly the whole wound was closed. On the third day, however, his temperature rose gradually to 104°, and he died without having recovered consciousness. The autopsy revealed nothing further than the condition disclosed at the operation, with the exception of a fine fissured fracture of the lesser wing of the sphenoid from which the orbital hæmorrhage was derived.

The points of clinical interest in this case, gentlemen, are of importance, and I will endeavour to make them clear. Meningeal hæmorrhage, as you are aware, is one of the tragedies of surgery. It is often insidious in its onset and dramatic in its culmination. It is one of the pitfalls of hospital and at times of private practice, being frequently the origin of serious trouble when a patient is unfortunately discharged from the casualty room under the impression that he has been slightly concussed, only to be brought back later on, dying or dead in an ambulance. Although more commonly seen in adults, and in them always associated with fracture of the skull, it is also met with in young children, and in them fracture may be absent, for so elastic is this portion of the calvarium in these patients that it is possible for a blow to so depress the skull momentarily that on the return of the latter to its normal position the dura-mater, always more loosely connected with it in young children, undergoes sudden and forcible detachment with rupture of one or even both branches of the artery. It is obvious, however, that meningeal hæmorrhage is very much more prone to occur in adults for the following reasons: firstly, the artery is more brittle in elderly and atheromatous subjects and in the victims of syphilis; and secondly, it at times runs for part of its course in an actual tunnel in the bone or in a groove so deep as to contain at least two thirds of the circumference of the vessel, passing from this on to the surface of the dura mater; in such cases a fracture is very apt to tear the vessel at this point.

The case before you illustrates very forcibly the clinical course of events. That is to say, an injury to the head, which causes temporary unconsciousness, followed by a period of recovery, the duration of which may vary from minutes to hours, and which ultimately gives way to drowsiness, coma, epileptiform convulsions of the opposite side, spasm and death. The mere fact of a return to unconsciousness in such cases should be sufficient to point the diagnosis, and to delay in waiting for any more confirmatory sign is to seal the patient's doom. Such confirmatory signs were not lacking in this case, and it is worth while to consider the consecutive order in which these commonly occur, since they are to some extent an indication of the extent of the lesion and of the prognosis.

Where the interval of consciousness is brief, the drowsiness irresistible, and the ensuing coma deep, the lesion may be expected to be extensive and the hæmorrhage rapid. In such cases you may at times observe epileptiform twitchings of the opposite side of the body even before coma sets in and while the patient is still conscious but drowsy. About the time at which coma sets in the pupillary changes become obvious.

Now, I am aware from experience that the exact cause and significance of these changes is apt to be vague in the minds of many students, and, there-

fore, I would put them before you briefly thus: The effect of intracranial pressure on the pupils is to cause first contraction and then dilatation, *but the pupil on the side of the injury is always the leader in the race.* Thus, in a right-sided hæmorrhage, it is the right pupil which first contracts, and by the time it is nearing pin-point the left pupil will have begun to follow its example; when the right stands in full dilatation, the left will also have begun to expand. You may, however, be faced by a difficulty here, when you have nothing to guide you as to which is the injured side; for in cases in which the difference in diameter is not very marked, you may fancy that the larger of the two is the normal which has not yet commenced to contract, and, therefore, that of the sound side, whereas it may actually be that which commenced first and has now reached three quarter dilatation and is that of the injured side. As an instance of this I was once present at a trephining in which a semi-dilated pupil suggested a hæmorrhage of that side: nothing was found there and the calvarium was opened on the other side, when a large effusion of blood was found. How can you avoid such a mistake? It may be impossible; but the following points may help you.

If twitching is still present in one hand, arm or leg, it will point to the injury being on the opposite side of the brain; if no twitching is present and the stertor is deep, if the spasm affects both sides pretty equally, and the pulse is full and bounding and the congestion marked, the case is obviously well advanced, and both pupils having reached pin-point and commenced to dilate, the larger should point to the side of injury.

As regards prognosis from these eye changes, it may be said that when one pupil stands in full dilatation and the other has commenced to dilate, the prospects of recovery are distinctly small; they are much more favourable if the case can be dealt with before the second pupil reaches pin-point. In the case I have operated upon the prognosis was bad since (1) within fifteen minutes of the recurrence of coma both pupils had dilated, that on the side of injury being in full expansion; (2) spasm was present on both sides, and convulsive movements had ceased; and (3) the external evidence of injury was extensive.

On the other hand, it was just possible to hope for success for four reasons, viz. that his pulse was still forcible and not running, his spasm had not passed into paralysis, stertorous breathing, although present, was not very marked, and his coma had not existed very long.

Cases with such a clear history as the present then, gentlemen, present no great difficulties of diagnosis, but it is quite otherwise with those in which the patient comes to your notice without any history to help you other than that he has been found unconscious, and without any external evidence of injury.

A diagnosis must be made, and that early, since all conditions resulting in coma are serious, and some may be treated with success. Five conditions will come into your mind as possible causes. They are:

(1) Intracranial hæmorrhage, *i.e.* meningeal, cerebral, or basal.
(2) Uræmia.
(3) Diabetes.
(4) Narcotic poisoning.
(5) Alcoholism.

Let us consider them in turn. Meningeal hæmorrhage has already been dealt with. Cerebral hæmorrhage (*i.e.* into the basal ganglia, usually from the lenticulo-striate artery) is seen especially in elderly atheromatous or syphilitic subjects. It is commonly very rapid, coma coming on from the first, and there being no return to consciousness; epileptiform convulsions and spasm are absent; the pupils are commonly equal, and show some dilation. Stertor is very marked, and the face is deeply congested. It is quite clear from the first, that even if the case is taken for one of meningeal hæmorrhage, the flaccidity of the limbs points to the last stage of the trouble, and therefore operation is out of the question. The same remarks hold good with regard to basal hæmorrhage, and it is, of course, rare to find an extensive basal hæmorrhage without some other sign of fracture, such as the appearance of blood, and possibly cerebro-spinal fluid, from the auditory meatus, nose or mouth.

In the case of uræmia assistance will be obtained by examination of the urine, and this examination should never be omitted in these cases of coma, although I fear it is frequently overlooked in the hurry of the moment. Epileptiform convulsions are more likely to be met with in the later stages of the case, and are bilateral from the first. The

pupils are similar in size and *contracted*—an important point, since at only one period in meningeal hæmorrhage are they similar in size, *i. e.* in the very last stage, when both are widely dilated.

The coma of diabetes is hardly likely to mislead you, since the respiration is rather sighing than stertorous, the face lacks the marked congestion of intra-cranial hæmorrhage, while a close examination may lead you to find evidence of pruritus, furunculosis, or the odour of acetone in the breath or sweat, and again the urine will confirm your diagnosis provided you remember to examine it.

Narcotic poisons do not give unequal pupils, muscular spasm, epileptiform convulsions, stertorous breathing, nor facial contraction. Opium and its alkaloids, as you are aware, induce three well-known effects, viz. pin-point pupils (which are equal), lividity of mucous membranes associated with facial pallor, and feeble fluttering pulse. The odour of the breath is characteristic, especially if the liquid preparations have been taken. None of the narcotics give rise to the active stertor and congested facies of intracranial hæmorrhage. Alcoholic poisoning, on the other hand, so far as respiration, pulse and congestion are concerned, may clearly simulate this condition; the pupils, however, are always equal, usually in mid-dilatation, and the whole body is completely flaccid. The odour of the breath cannot be relied upon to help you here, since intracranial hæmorrhage may have resulted from a debauch, or the patient may have been heavily dosed by unthinking friends as a remedy for his coma.

Look at it as you will, gentlemen, the diagnosis between these five conditions is often difficult, and at any time an error may brand you quite undeservedly with the stigma of having mistaken an intracranial hæmorrhage for an alcoholic poisoning, washed out the victim's stomach and consigned him to the care of a well-meaning but unsympathetic police.

Now a word or two as to the technique of operation in meningeal hæmorrhage. There must be no waste of time; the condition is so urgent that if it were possible it would be better to open the skull where the patient lies, be it bed, ambulance, or floor. This is a point which few nurses seem able to realise, and often much delay occurs owing to a laudable desire to shave the head, to array the patient in fair white linen, to "clean him up," or to observe the Draconian ritual of a soap-and-water enema. Speed in such cases must even replace asepsis if necessary, nor must you allow yourselves to be hampered by anatomical minutiæ in the finding of the correct point at which to trephine. There are three essentials in this operation and I would have you remember them: firstly, cut a large flap; secondly, make a big hole; and thirdly, do it rapidly. You may use a trephine, the largest you have, to commence with, but you will make more progress with Lane's or Hoffmann's forceps when once you have entered the skull. This done, you will turn out the clot with your finger or a scoop and turn your attention to securing the meningeal artery. It is not always an easy matter if the hæmorrhage is brisk and the injury low down. If by good luck you succeed, as I did in this case, in seizing the vessel with forceps, do not attempt to ligate it if the point of the forceps is out of sight; you will probably fail, and you may not succeed a second time in catching it. If you do not think your patient will stand any more extended operation, you will be wise to leave the forceps in position for forty-eight hours, supporting them carefully with the dressings. One of you has asked me if I would not resort to plugging when the vessel could not be reached. I would give a most emphatic reply in the negative. Plugging can only control hæmorrhage when it can be applied firmly to the bleeding point; the plug requires a "*point d'appui*"; if such is to be obtained within the skull, it can only be at the cost of its soft contents, and what object can there be in relieving the pressure of clot if you are to replace it by that of gauze? I have seen attempts made in three different cases; the result was invariably the same: the hæmorrhage went on and the patients died. If I could never impress anything else upon you, gentlemen, I would like to impress upon you the futility of such treatment. The mere fact that the patient's life depends on the speedy expansion of the brain to its normal capacity should be sufficient argument against plugging. Very occasionally it may be possible to plug the vessel with aseptic wax, but this can only be done when it lies in a definite tunnel in the bone—a very rare occurrence. When the vessel can neither be caught nor plugged, there remains only ligation of the external carotid artery, and my advice to those of you who may

have at any time to perform this operation, and who may not have had considerable experience of neck surgery, would be not to waste precious time in attempting what may prove a difficult matter, especially in patients with stout necks, but to essay the less troublesome operation of tying the common carotid above the omohyoid muscle. The flap of scalp should then be lightly closed, a small wick of gauze being left in the posterior angle of the wound for drainage, and this may be removed at the end of forty-eight hours.

The prognosis will depend largely on five factors, viz. (1) lapse of time between the onset of coma and the clearing out of the clot; (2) the degree of flattening of the cerebral convolutions; (3) the rapidity of expansion of the brain on removal of the clot; (4) the completeness of the reaction to the release of pressure; and (5) the absence of septic complications. Death, when it follows operation, is commonly due to one of three conditions, viz. (1) inability of the brain to expand, the patient's condition of coma remaining unchanged, his respiration and circulation becoming finally paralysed; (2) disorganisation of the pyrexial centre, his temperature rising steadily to 106–107° F.; or (3) to the supervention of intracranial sepsis, in the form of meningitis or encephalitis.

September 1st, 1913.

Soda Dropsy.—Breitmann noticed that œdema developed suddenly in a diabetic boy given large doses of alkali for acidosis. The elimination of sodium chloride in this and a number of similar cases was apparently normal, but the children increased in weight and became almost transparent from the retained fluids. The course of the cases showed that this œdema was an unfavourable sign. He had similar experiences with this œdema following administration of an alkali in suppurative cystitis, chronic intestinal trouble, in lordotic albuminuria, in gout, and with septic processes. As it is frequently important to give an alkali, its dropsy-producing action must be counteracted by some other drug. His results have been encouraging with sodium acetate or injection of pilocarpine. Belladonna preparations are contra-indicated as they reduce glandular secretion. He warns particularly against permitting this soda dropsy in patients with acidosis or the lymphatic constitution. It may occur with intact kidneys. It is a question whether the retention of fluids is induced by the alkali itself or indirectly by its combination in the form of sodium chloride.—*Journ. A.M.A.*, vol. lxi, p. 318.

ON THE LATENCY OF SYMPTOMS IN FRACTURE OF THE NECK OF THE FEMUR IN ADOLESCENTS.*

By E. D. TELFORD, F.R.C.S.,
Assistant Surgeon, Manchester Royal Infirmary.

I HAVE had under observation during the last three months two cases of this injury which are of interest as examples of a lesion by no means rare, and one which may easily lead to error in diagnosis. The two cases present a remarkable similarity.

(1) A boy, æt. 17 years, farm labourer, of good muscular development and general physique. Three months before he came under observation he fell on the left hip whilst running; the hip was stiff after the fall, but he went on with his work, and had no treatment. At the end of three months he was brought complaining of increasing pain and stiffness of the left hip. The condition found on examination was as follows: The left lower extremity was everted, and showed one half inch of real shortening; the trochanter was raised, and there was much wasting of muscle, especially in the thigh. Flexion of the hip was limited to one half its normal range; abduction and adduction were much limited, and rotation of the hip almost abolished. A radiogram showed a complete fracture of the neck of the femur at its insertion into the shaft. The angle between the line of the neck and the shaft was almost a right angle.

(2) Male, æt. 15 years, farm labourer, of good health and physique. This boy fell heavily on his right hip twelve months ago, but went on with his work for three months, at the end of which time the increasing pain and stiffness led to his being treated for tuberculous disease of the hip. I saw him twelve months after the injury. There was then slight eversion of the lower limb, with one half inch of real shortening, a raised trochanter and marked wasting of the thigh-muscles. All movements of the hip were possible to one half of their normal range. A radiogram showed a fracture through the intertrochanteric line, with the neck of the femur at a right angle to the shaft,

* Abstract of a Clinical Lecture delivered at the Manchester Royal Infirmary.

the line of fracture showing an excessive amount of callus.

Previously to the discovery of the X rays there are but scant references to the injuries of the upper end of the femur in childhood, and, in fact, the subject can scarcely be said to have existed. These injuries are commonly dismissed by writers as conditions of great rarity, and such cases as are mentioned are tacitly assumed, mainly on the analogy of like injuries in the neighbourhood of other joints, to have been examples of epiphysial separation. The extended use of the X rays shows that gross traumatic lesions of the upper end of the femur in childhood are by no means so rare as had been supposed; and this is no matter for surprise when one considers the frequent and heavy falls of children, which throw so great a strain on a structure such as the femoral neck, composed as it is of soft growing bone, and further weakened by the presence of an epiphysial line. It is no wonder that the neck of the femur should not infrequently prove unequal to the strain.

The inspection of a number of radiograms, illustrating injuries of the hip in childhood, shows that the subject is to be divided into two sections. They are: (1) The separated epiphysis, (2) the fracture of the neck. Much of the confusion of the earlier writings is due to the failure to recognise this distinction, and for this reason it is not easy to estimate the relative frequency of the two types of injury. It is, however, probable that the fracture of the neck is at least as common as the separation of the epiphysis.

The fracture of the neck of the femur in children has been most commonly seen at the ages of ten to fifteen. The cause is usually a fall, which may be of no great violence. The injury appears to be more easily produced when the fall occurs with the limb in an abducted position. The line of fracture is commonly placed well to the outer side of the epiphysial line, and in a number of radiograms which I have examined it can be seen to run transversely across the neck at a point nearer to the shaft than to the head, and in some cases appears to follow the anterior intertrochanteric line.

After the fracture of the femoral neck, the course of the case will follow one of two types. In the one type the symptoms are at once obvious, but in the second they are singularly latent, and have often failed to attract attention until some weeks or months have elapsed and the accident is forgotten or remembered only with difficulty. In the former type there is an immediate displacement of the femoral shaft upwards on the neck, with outward rotation of the limb, and in these cases some gross lesion of the femoral neck is suggested at once by the position of the limb and the loss of function following immediately on a fall.

I am, however, at present concerned solely with the second type, in which the symptoms are long delayed. The explanation of this delay is to be found in the fact that the original fracture is either impacted or incomplete.

It is stated by some writers that impaction does not occur in these fractures, but I think it likely that impaction does occur in these cases. Many of the radiograms of these injuries in children strongly suggest that impaction has taken place, since they show plainly so complete a fracture through the neck that it is hardly conceivable that so grave a solution of continuity could have escaped notice, or that the patient could have walked after the injury unless the fracture had been firmly impacted. It is to be noted, further, that the large amount of callus surrounding the fracture suggests that there had been considerable dislodgment of the parts immediately after the accident. A further argument in favour of the impaction of this fracture in children is afforded by the fact that the similar fracture in adults is known to be commonly impacted. There is no doubt that some of these fractures are of the incomplete variety, and that the "greenstick" injury produces a weakening of the bone with subsequent bending of the damaged part.

The history of these cases in which the symptoms are delayed is usually as follows:

The patient, more commonly a boy, is brought on account of having of late developed a limping gait. On examination, there is noted muscular wasting of the thigh, with some half inch of real shortening of the affected limb, which presents a decided eversion and shows a trochanter raised above the normal position. The movements of the hip-joint are found to be limited in all directions, especially in the direction of abduction. A radiogram shows that the upper end of the femur is deformed, and that the angle between the neck and shaft is diminished in consequence of an

obvious crack across the neck, usually close to its insertion into the shaft. On close inquiry history of a fall may be elicited, and this accident has usually occurred at a time remote from the examination by at least three months. The explanation of the case is that the femoral neck was broken by the fall; the fracture was either "green-stick" or impacted, and the child continued up and about until the yielding of the damaged bone induced symptoms which are no longer those of fracture, but those of traumatic coxa vara.

There can be no doubt that these cases have not infrequently passed for cases of commencing tuberculous disease of the hip. One of my cases had already had three months' treatment based on this diagnosis. There is, of course, a considerable likeness in the two conditions. In either case a child is found to be limping, and on examination there are wasting, eversion, and limitation of movement. There is also complaint of pain, and the diagnosis is further complicated by a history of injury, commonly of so slight a degree that it suggests not a fracture, but merely a reason for the onset of tuberculous disease. If the treatment is carried out on a mistaken diagnosis of tuberculous disease the case will, of course, "recover," and the resultant disability of the hip is regarded as the natural result of healed tuberculous arthritis. The differential diagnosis of these traumatic cases from cases of true tuberculous disease is based on the fact that there is no flexion present, as is always the case in arthritis of the hip; that there is some shortening from the first; and that the patient may be a child whose family history, personal physique and appearance suggest that the possibility of a tuberculous lesion is at least remote. The true nature of the case is usually at once apparent if a radiogram be taken. One cannot, in fact, lay too much stress upon the necessity for radiographic examination in every case of suspected tuberculous arthritis of the hip.

September 1st, 1913.

Post-diphtheritic Facial Paralysis.—S. Wolff has described the case of a girl, æt. 3 months, who developed complete right facial paralysis three weeks after an attack of nasal diphtheria. 12,000 units of antitoxin were injected and recovery took place in three weeks.—*Brit. Journ. of Children's Diseases*, vol. x, p. 367.

A CLINICAL LECTURE

ON

A CASE OF CHRONIC NEPHRITIS.

By H. L. McKISACK, M.D., M.R.C.P.,
Physician to the Royal Victoria Hospital, Belfast.

THE patient we are about to examine to-day was brought into hospital a few days ago, suffering from difficulty in breathing, weakness, palpitation of the heart and vomiting. He is æt. 18 years, and is an apprentice plumber; hitherto he has led a healthy and regular life, free from any illness that he can remember. Without being especially athletic he has had a fair amount of open-air exercise, both as a boy and as a young man. Eighteen months ago he began to suffer from headaches and vomiting, which have recurred occasionally since; of late they have been more severe and frequent.

On admission to hospital his temperature was normal, his tongue was slightly coated, his pulse was frequent (108), fairly regular and of high tension. His aspect indicated a serious illness—his face was pale and slightly cyanotic, there was no œdema, but his breathing was somewhat laboured. His heart was found to be hypertrophied, particularly on the left side, and there was a loud systolic murmur best heard at the apex; at the base of the heart one heard a harsh, almost grating double murmur, synchronous with the first and second sounds of the heart. In the same region the second sound was unusually intense, and was especially loud and ringing in the neighbourhood of the second right costal cartilage. At the lower end of the sternum there was a systolic murmur apparently distinct from that at the apex. In the neck the internal and external jugular veins were seen to pulsate. The urine was found to be rather more copious in quantity than normal, its specific gravity was 1010, it contained 0·2 per cent. of albumen and 1·1 per cent. of urea; granular casts in considerable numbers, granular *débris*, and a few red blood-cells were found in the urine. His weakness has never reached the point of syncope, and he had never had any fit or convulsion.

Now, after a few days' treatment, as you will find on examining the patient, there is some improve-

ment in the condition of the heart, but otherwise the present state is very much as just described.

Previous to his arrival at hospital he had not, so far as I am aware, received any medical treatment, and he came here to seek advice and aid for what his friends had diagnosed as disease of the heart.

It is possible that the symptoms and history may seem to some of you suggestive of mitral valve disease. It might be argued that the patient had been suffering from incompetence of the mitral orifice, as indicated by the hypertrophy of the left side of the heart and the murmur characteristic of mitral regurgitation, with passive congestion of the kidneys as a consequence of that defect in the circulation. To this diagnosis there is the unanswerable objection that the pulse is of high tension, that the second sound is most accentuated on the *right* side of the sternum, and that the urine contains a considerable quantity of broken-down kidney-cells and casts. Further, the evidences of defective circulation are by no means commensurate with the gravity of his condition; thus there are no bronchial catarrh, hæmoptysis, or other signs of increased pulmonary tension; there is no œdema; there is no serious obstruction to the return of blood from the portal circuit.

On the other hand, the picture of chronic nephritis in an advanced stage is complete: he has headache, he sleeps badly, he vomits—indications of inefficiency of the kidney which fails to remove from the circulation the necessary amount of waste and deleterious substances. This inactivity of the kidney is even more clearly shown by the reduced output of urea, which, in spite of a somewhat copious secretion of urine, is only a little more than half the normal quantity, and the specific gravity of the urine is consequently lower than normal. The pulse-tension is raised, as you will verify by digital and instrumental examination of the radial artery, and by listening over the second right costal cartilage to the accentuated second sound. The ventricles are enlarged, both being hypertrophied and dilated. It is this dilatation of the ventricles which, by preventing the accurate apposition of the cusps of the valves, permits a reflux through the mitral and tricuspid orifices, thereby producing the systolic murmurs in the mitral and tricuspid areas and the venous pulsation in the neck. The rough double murmur heard in the neighbourhood of the second left costal cartilage is less easy to explain; it may, in spite of the absence of præcordial pain, be an evidence of pericarditis, a not infrequent result of Bright's disease; its quality or character, and the character of the pulse are strong arguments against its being of aortic origin.

It can, then, be confidently asserted that the very serious heart mischief from which this patient was obviously suffering on his admission to hospital, and which to a less degree still oppresses him, is the result, not of an endocardial valvular lesion, but of myocardial degenerative changes, the result of chronic disease of the kidneys.

Let us look closer into the state of his kidneys, so that we may determine, if possible, the exact nature of the affection. With the diminished output of urea and the presence of granular casts and *débris* of kidney elements, we have no difficulty in locating the mischief in the kidneys themselves, and not in the urinary passages lower down. New growths and tuberculosis, in the absence of appropriate symptoms, may be excluded. We may, then, assume that we have to do with either chronic inflammation of the kidneys (chronic Bright's disease), or the chronic renal arterial sclerosis known by the various names of gouty, contracted, or small red kidney. Some degree of probability is lent to the last named condition by the fact that the patient in his occupation has been exposed to the influence of lead, which is known to be a causal factor in the production of the small red kidney; also the quantity of urine passed being larger than normal would suggest this affection. On the other hand, the fairly large amount of albumen and the numerous granular casts enable us to reject the diagnosis of the small red or arterio-sclerotic kidney.

We are thus narrowed down to chronic Bright's disease (for we may safely exclude the amyloid kidney in the absence of its usual causes), and we have now only to decide which form of lesion is present. That is, have we the large white kidney (chronic parenchymatous nephritis) or the small white kidney (chronic interstitial nephritis)? The absence of œdema and the ample secretion of urine argue against the large white kidney, while these two conditions of the urine, together with the character and amount of casts, the quantity of albumen, the low specific gravity of the fluid, and the high pulse tension, are consistent with a moderate

degree of interstitial nephritis. It is, therefore, probable that the patient's condition has been brought about by a chronic interstitial nephritis, which may possibly have been parenchymatous at the commencement, but which now has arrived at a fair degree of sclerotic or cicatricial change.

Can we hope that this patient will be restored to health by treatment? Provided that his kidneys and other excretory organs can be induced to act more efficiently in removing harmful waste products of metabolism from the blood, the heart may regain sufficient propulsive power to carry on its work with safety for a time.

It is to facilitate the removal of waste from the blood that our treatment is primarily directed; the bowels must be made to act freely, the skin must perspire profusely, and the quantity of urine must, if possible, be increased. At the same time we must actively treat the weakened heart muscle with strychnia and other heart tonics, preventing the expenditure of any needless energy by strict rest in bed.

P.S.—The subsequent course of this case was disappointing. After making a decided improvement as to the condition of the heart, the kidneys became less efficient with a still further diminished output of urea, in spite of the administration of diuretic and diaphoretic drugs and suitable feeding. Three weeks after admission his uræmic symptoms deepened into actual convulsions and coma, and he died six days later. At the post-mortem examination the heart was hypertrophied and dilated, the tricuspid orifice admitted four fingers, and the mitral orifice was also enlarged; the kidneys were rather smaller than normal, the capsule was adherent, the surface granular, the cortex paler than normal and reduced in thickness, the medulla dark red. No other lesions of importance were found. The rubbing murmur heard at the base of the heart was unexplained.

September 1st, 1913.

Necrosis of the Jaw following Measles.—J. A. Graham has reported a case of this complication in an infant æt. 11 months.—*Brit. Journ. of Children's Diseases*, vol. x, p. 370.

A Text-Book of General Pathology. By various Contributors. Edited by M. S. Pembrey, Lecturer in Physiology, Guy's Hospital, and J. Ritchie, Superintendent, Royal College of Physicians' Laboratory, Edinburgh. 773 pages. London: Edward Arnold. 15*s.* net.

This book is intended for the use of advanced students and practitioners. The introduction, ably written, is the joint production of the editors. Health, from the subjective point of view, is regarded as "the capacity for passing with a sense of well-being through a constantly recurring cycle of activity and rest." Disease is stated "not to depend on the development of any new properties in protoplasm, but rather to result from an interference in the correlation between the different activities of living matter which constitute the state of health."

The chapter on Inflammation is by E. W. Ainley Walker, so is that on Infection, including the mode of infective agents. Immunity is in the hands of C. H. Browning; Degeneration, Necrosis and Calcification are considered by Lorrain Smith. Chapter VIII, on Tumours, by Ritchie, justifies and will amply repay careful reading. In regard to age-incidence of these, he states that cancerous tumours are most frequent after forty-five and excessively rare before twenty. Sarcoma, though more common than cancer in early life, is most frequent after thirty-five; a feature of the latter growth is the relative frequency during the first five years of life.

The chapter on the Pathology of the Circulation is by A. E. Boycott. Here in one article is admirably epitomised information, for which one has often to wade through different authorities. The pathological entities are discussed with an ease and freedom which admit of easy assimilation, and which no wise practitioner will omit. The Nervous System is discussed by Mott and Sewell. Haldane writes on Respiration—a subject peculiarly his own. Temperature is from the pen of Pembrey, so is Metabolism. Hertz is responsible for the "Pathology of Digestion," Garrod for Metabolic Disturbances from Hepatic Disease and for Anomalies of Metabolism. Beddard writes on Diabetes, Walker Hall on Gout, and Ryffel on the Kidney. The volume fitly concludes with an article on Death by Ritchie.

This is a book no practitioner who wishes to be up to date can dispense with.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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ACUTE ABDOMINAL PAIN IN PREGNANCY.*

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IN considering the subject of "Acute Abdominal Pain in Pregnancy," I propose to deal only with conditions which I have met with myself. These comprise extra-uterine pregnancy, extra-uterine complicating intra-uterine pregnancy, molar pregnancies, acute hydramnios, pyelonephritis, appendicitis, twisted ovarian tumours, intra-peritoneal adhesions, whether due to previous inflammation or to ventral fixation, degeneration of uterine fibroids, constipation with more or less acute intestinal distension, concealed accidental hæmorrhage, and pneumonia and pleurisy. If we do not get through the whole list we ought to find enough material to produce some discussion, and probably some of you will be able to bring forward other important and interesting conditions which I have omitted to mention. I might extend the list *ad nauseam* if I did not confine myself to my own experience, and to cases in which the diagnosis affords points of interest. The word "acute" cuts out a certain number of conditions. I should like to preface my remarks by the warning that it is not safe to consider, as some men are inclined to do, that because a woman is pregnant and has acute abdominal pain, therefore the pregnant uterus itself is responsible for the pain. I believe that a good many cases of, *e. g.*, pyelonephritis of pregnancy are missed and the patient condemned to a great deal of unnecessary suffering because the pain is diagnosed as being due to "painful uterine contractions," and treated only with sedatives. There are some cases in which the uterine contractions which occur during pregnancy cause more or less

* Read before the London Hospital Medical Society.

distressing pain, but these cases, which are not common, must be found out by a process of exclusion.

Conditions in which the uterine wall itself is tender are not common. They include cases of concealed accidental hæmorrhage, degenerated fibroids, some cases where ventral fixation has been performed, and comparatively rare cases of adhesions due to pelvic inflammation. Besides these must be mentioned cases in which the fœtus is unusually lively, and seems to kick with great pertinacity at one spot, until the unfortunate patient says that she quite expects to find her abdominal wall "black and blue." These last, however, can hardly be included in the term "acute abdominal pain." A good deal of relief may be experienced by the use of an abdominal binder.

Pyelonephritis of Pregnancy.

Pyelitis or pyelonephritis of pregnancy is a condition that has been recognised only recently, and some text-books published within the last seven years omit any reference to it. In 1892—twenty years ago—Rebland reported six cases. During the last ten years a large number of papers on the subject has been written, and many cases have been recorded, and it is gradually being recognised that the condition is a common one. Although not often causing danger to life, the condition may cause so much pain and impairment of health that its early recognition and treatment are important.

Symptoms and physical signs.—The symptoms usually begin during the second half of pregnancy, and vary greatly in severity. In some cases there is only aching in the lumbar regions, with no constitutional disturbance. I believe that cases in which there are no acute symptoms are comparatively common. I used to see them fairly often in the out-patient department. If they are unrecognised, and, therefore, untreated, the patient undergoes a considerable amount of pain and discomfort which might be relieved fairly easily. The symptoms in more severe cases are pain in the loins and lower abdomen, sometimes very severe, with acute exacerbations, suggesting renal colic, and accompanied by vomiting. The pain may be so severe that the patient's sleep is interfered with to a serious extent. It may be impossible to say whether the affected kidney is enlarged or not, the presence of the pregnant uterus rendering palpation of the kidney regions difficult, but tenderness of one or both kidneys and tenderness along the course of the ureters is found on pressure. In a case in "Victor" recently the right kidney could be palpated easily, enlarged to at least twice its normal size, the patient being about six months pregnant. In some cases there is little impairment of general health and nutrition; in others the patient is obviously very ill. The temperature is sometimes normal, but usually raised, and there may be a high degree of pyrexia, usually with oscillations, and rigors may occur. The pulse-rate is, of course, rapid when there is much pyrexia. On vaginal examination the intra-pelvic portion of the ureter may be found to be tender, and if there is cystitis the base of the bladder is tender to the touch. Micturition may be frequent and painful. The urine is usually acid, and on superficial examination, even when severe symptoms are present, it may show little that is abnormal. After it has been allowed to stand for some time, however, or after centrifugalisation, a deposit of pus is found. Sometimes there is a very large amount of pus, but pus may be almost or entirely absent on a single examination. It is possible that the occasional absence of pus may be accounted for by temporary blocking of the uterer on the affected side. Bacteriological examination reveals the presence of micro-organisms, most commonly the *Bacillus coli communis* or a "coliform" bacillus. Recurrence in subsequent pregnancies is common.

Diagnosis.—From consideration of the symptoms it will be seen that diagnosis of the condition may be simple and easy. In other cases it may be difficult, usually, however, because pyelonephritis is not thought of. Suspicion of the presence of pyelonephritis should always be raised by complaint of pain in the abdomen, loins or back, or by pyrexia during pregnancy. If the pain and tenderness are confined to the right side appendicitis may be wrongly diagnosed. I have known a case in which the appendix was removed before the correct diagnosis was made. Sometimes enteric fever has been diagnosed, also other gastro-intestinal troubles and influenza. In some cases which were at first thought to be pyelonephritis of pregnancy it has been proved later that a renal calculus was the cause of the trouble. One occasionally sees a pregnant woman who seems to be almost

well, with only slight rise of temperature and pulse-rate, twenty-four hours after a rigor with high fever. This is very suggestive of pyelonephritis. As a rule careful abdominal and vaginal examination, and examination of the urine, if necessary on more than one occasion, including bacteriological examination, will leave no doubt as to the correct diagnosis.

Ætiology.—There is no consensus of opinion as to the method of infection of the kidney. The most obvious suggestion is that there is an ascending infection from the bladder—that is, a cysto-ureteropyelonephritis—and there are a good many points in favour of this view.

Cystitis is certainly more common in women than in men, and often exists in pregnant women without its presence being suspected, frequency of micturition and discomfort being explained as results of pregnancy.

Dilatation of the ureters undoubtedly occurs fairly often in pregnancy, as is proved in post-mortem examinations of cases of eclampsia, and when present will favour ascent of micro-organisms from the bladder. This dilatation may be caused by the pressure of the uterus at the pelvic brim. The right ureter is more commonly pressed on by the uterus than the left, as it lies rather farther from the middle line, and so gets less protection from the projecting promontory of the sacrum.

Opitz looks on the lower part of the dilated ureter as containing a stagnant column of urine. He quotes experiments of Guyon and Albarran on animals, showing that when a stagnant column of urine is present in the ureter there is really a free communication between the bladder and the ureter, so that organisms can pass upwards without any obstruction being offered by the valve-like opening of the ureter.

However, if dilatation of the ureter is accepted as a factor in the development of pyelonephritis, it is not necessary to postulate the presence of a column of stagnant urine to explain the ascent of micro-organisms from the inflamed bladder to the kidney. The researches of Bond, of Leicester, recorded a few years ago at the annual meeting of the British Medical Association, show that micro-organisms may possibly travel along the mucous membrane of the ureter against the stream.

The main objection to this obvious suggestion, that the cause of pyelonephritis of pregnancy is ascent of micro-organisms from an inflamed bladder along a dilated ureter to the kidney, is that in the majority of cases there is no cystitis. Many observers are agreed on this point while admitting that pyelonephritis occurring during the puerperium may arise from cystitis.

The explanation which receives most support accepts the fact that there is some dilatation of the ureter and pelvis of the kidney, but attributes the origin of the micro-organisms to the blood or bowel rather than to the bladder. The renal epithelium is damaged partly as the result of increased pressure from below and partly from toxæmia of pregnancy—that is, its resistance is lowered.

The frequent occurrence of constipation during pregnancy may explain the entrance of the *Bacillus coli communis* into the pelvis of the kidney. The close relation of the ascending colon, often containing an accumulation of fæces, to the right kidney is held by Mirabeau to account for the frequency of right pyelonephritis. Cumston suggests that there may be an exalted virulence of the colon bacillus due to some gastro-intestinal disturbance.

Treatment.—Almost all writers on the subject are in agreement that the treatment of pyelonephritis of pregnancy should be as conservative as possible. The general treatment consists of rest in bed, milk diet with plenty of harmless fluids to drink, and regulation of the action of the bowels. Violent purgation must be avoided as being likely to cause miscarriage. Some authorities advise administration of small doses of calomel, *e. g.* gr. $\frac{1}{6}$ *t.d.s.* I never order repeated small doses of calomel now, as I used to find that they caused a good deal of colic. More special treatment includes administration of diuretics, and urinary antiseptics, alteration of the reaction of the urine, etc. Diuresis is secured by administration of infusion of buchu, and sometimes by digitalis. Several writers mention distension of the bladder as causing a reflex diuresis. I have no experience of this method of treatment. The most popular urinary antiseptic is urotropin, gr. xv–xxx of which may be given daily. As the organisms are growing in an acid medium the urine should be rendered alkaline or neutral. This is done by administration of citrate of potash, gr. xx–xxx *t.d.s.*, or of alkaline waters. Antitoxin treatment has been tried with good results (Dudgeon, quoted by Thomson Walker). Results of vaccine

treatment are disappointing, at any rate in acute cases. Pain is treated by warm applications to the loins, and if necessary to the abdomen, by administration of tincture of hyoscyamus, ℥xxx *t.d.s.*, and by posture. Sometimes great relief is obtained by having the foot of the bed raised one or two feet, while in other cases a sitting posture may be found to give relief. As a rule the pain soon yields to the treatment mentioned above. Interruption of the pregnancy should be avoided if possible, expectant treatment always being given a fair trial. Some writers emphasise the increased risk of puerperal infection on account of the septicity of the urine, but in Legueu's 52 cases there was no puerperal sepsis, and there has been no sepsis in cases delivered in the London Hospital. If both kidneys are affected, and the condition does not yield to medical treatment, and the patient's general condition is going from bad to worse, it may be necessary to empty the uterus, but it would be better, as a rule, to operate on the kidney than to empty the uterus.

The following is a typical case.

H. A—, æt. 32 years, admitted in July, 1912, complaining of severe pain for one month, intermittent at first, continuous for the last two weeks in the right side of the abdomen running down into the groin, not much pain in the back. Married ten years, had three children, the last fifteen months ago; no trouble with any of her previous pregnancies. The patient was found to be five months pregnant. The right kidney was enlarged and tender. The temperature on the first two nights was 101·5° F., pulse 98 to 114; the urine was acid, and contained pus in which the *Bacillus coli communis* was found. She was kept in bed and given a diet consisting almost entirely of milk. On the fifth day the temperature became normal, and remained so; the pain all went, although the urine still contained a small quantity of pus. She was admitted again in October, 1912. There was a large quantity of pus in the urine, and the *Bacillus coli communis* was found again. She had severe pain all over the abdomen. She had now tenderness in the left loin as well as the right. She was given a milk diet, and an alkaline mixture containing sodium citrate and sodium bicarbonate. The pain was greatly relieved by the foot of the bed being raised, and by fomentations to the loin. There was very little rise of temperature: it was above 99° F. on two occasions only. Labour came on naturally three weeks after her admission to the hospital, and a living, full-time child was born. There was still a trace of pus in the urine when she was discharged.

Adhesions.

Acute abdominal pain due to adhesions pulling on the pregnant uterus is seldom seen except in cases where ventral fixation has been performed. If the uterus itself is stitched to the abdominal wall, severe pain may be caused in subsequent pregnancy. I have not had to open the abdomen myself to free the uterus, but many operators have found that the pain was sufficiently severe to warrant this proceeding. I have seen two cases in which the patient has complained of severe pain, in both of which the uterus had been stitched directly to the abdominal wall. In the first one the patient described the pain as agonising, but she was able to perform her ordinary household duties, and her sleep was not interfered with. She looked extremely well and had an uneventful labour at full time. The other one was an unusual case. I saw her when she was nearly two months pregnant; the cervix was held up by the ventral fixation, but the body of the uterus was acutely retroflexed, the fundus being low down in Douglas's pouch. I pushed it out of Douglas's pouch, but could not get it into the normal position as the cervix was held upwards and forwards, and inserted a rubber ring pessary which prevented it coming down again. I heard later that she was successfully delivered at full time. When I have fixed the uterus itself to the abdominal wall in a patient who may possibly become pregnant again, I have always sutured the uterus as low down as possible, that is, just above the reflection of the vesico-uterine peritoneum. If the uterus is very soft and able to become retroflexed, this method is not to be advised, as is seen by the case that I mentioned just now. I have never been able to understand the sense of suturing the posterior part of the fundus to the abdominal wall in a patient who may become pregnant again. If any part of the uterus except the lower part of the anterior wall becomes firmly attached to the abdominal wall, there may be very great difficulty in labour, the uterus developing almost entirely at the expense of the posterior wall and fundus, the anterior wall forming a thick, fixed swelling in the anterior part of the brim of the pelvis. Eight years

ago I collected records of 189 cases of pregnancy and labour after ventral fixation. Among these 189 there were three cases of rupture of the uterus, and sixteen in which Cæsarean section was performed. Interesting facts connected with the ventral fixation in these cases are that in some of them the fundus, sometimes even the posterior surface of it, had been attached to the abdominal wall; in some of them a large number of sutures had been used—in one case fourteen—in others silk-worm gut had been used, and in some of them suppuration had occurred. On account of the possibly bad effect of ventral fixation on subsequent pregnancy, many modern operators have gone back to fixation by means of the round ligaments, shortening them by an intra-peritoneal operation. The two operations in most use are: (1) The sling operation; (2) Gilliam's operation. In the sling operation a loop of round ligament is drawn through the lower part of the broad ligament on each side and stitched to the posterior surface of the uterus low down. In Gilliam's operation a loop of round ligament is brought up into the abdominal wall on each side external to the rectus muscle, and then sutured to the aponeurosis on the opposite side. I have performed each of these operations a good many times, but so far I have not any details with regard to their effect on subsequent pregnancy and labour.

Apart from the possible bad effect on subsequent pregnancy and labour, ventral fixation of the uterus, in which the uterus itself is stitched to the abdominal wall, has another drawback, viz. the possibility of strangulation of a coil of intestine by kinking produced by the band formed between the uterus and abdominal wall. The round ligament operations are supposed to obviate this danger, but that they do not invariably do so is proved by a case in which I had occasion to open the abdomen recently, a year after Gilliam's operation had been performed by another operator. I found that the right round ligament had been completely separated from its peritoneal investment for about an inch and a half, so that a hole was formed between the round ligament and pelvic wall through which a coil of intestine could easily have passed. If ventral fixation is performed, the danger of the occurrence of internal strangulation should be prevented by occlusion of the vesico-uterine pouch by sutures, making a sort of median antero-posterior diaphragm between the lower part of the uterus and the anterior abdominal wall.

Adhesions due to Old Pelvic Inflammation.

I have seen one or two cases in which pain of a dragging nature in early pregnancy has seemed to be due probably to pulling on adhesions left behind after a pelvic inflammation, but they were not very severe and passed off as the pregnancy advanced. It is probable that if the uterus is very adherent pregnancy will be impossible owing to closure of the abdominal ostia of the Fallopian tubes. If the adhesions are not so dense and binding as to prevent pregnancy they will probably become softened as the result of the greatly increased blood-supply accompanying pregnancy, and will cause little trouble.

Extra-uterine Pregnancy.

In cases of extra-uterine pregnancy the presence of pain is a characteristic feature. In some of them it may be due to painful contractions of the uterus, but in the majority of cases it is due to bleeding either into the peritoneal cavity itself or into an encysted collection of blood. When fresh bleeding into a walled-off hæmatocele takes place the pain is sometimes exceedingly severe. In a case where the formation of a mole in the tube is suspected and expectant treatment is being tried, the occurrence of severe abdominal pain is an important indication for operation. Extra-uterine pregnancy is so familiar to most of you that I need say little about it to-night, except to emphasise the fact that the occurrence of slight external bleeding accompanied by pain in early pregnancy must always arouse suspicion that the ovum is occupying an abnormal site. I have recently had under my care two cases of combined intra- and extra-uterine pregnancy. In both intra-peritoneal bleeding occurred. In one, of which I will read some details, the intra-uterine ovum was expelled before the removal of the extra-uterine gestation sac. In the other the intra-uterine pregnancy continued undisturbed, in spite of abdominal section and removal of the pregnant tube and a good deal of blood-clot.

Mistakes in diagnosis most commonly made in cases of severe internal hæmorrhage due to tubal pregnancy are those of attributing the condition to perforation of the appendix, perforation of a gastric or duodenal ulcer or to twisting of the pedicle of

an ovarian tumour. The physical signs in all these conditions may be more or less alike. There is, however, no evidence of loss of a large quantity of blood in any of the last three conditions, and even if there has been no period of amenorrhœa to point to a correct diagnosis, a slight loss of blood from the uterus ought, as a rule, to suggest it. In one week I operated on two cases, one of severe internal hæmorrhage due to tubal pregnancy, the other of acute torsion of the pedicle of a large, solid ovarian tumour, with a considerable amount of ascitic fluid. The diagnosis with which the patients had been sent into hospital was wrong in both cases, the twisted tumour case being sent in as one of ruptured tubal pregnancy, the tubal pregnancy as one of twisted tumour. There was no difficulty in making a correct diagnosis before operation in each case. If, however, a vessel in the wall of a twisted ovarian tumour gives way so that bleeding occurs into the abdominal cavity, or there is a good deal of ascitic fluid present or a thin-walled cyst ruptures after twisting of its pedicle, the diagnosis may be difficult. It is rare for a twisted ovarian tumour to be too small to be felt on careful bimanual examition, if not on abdominal examination, except in some cases where the patient is pregnant. The fact that a definite tumour apart from the uterus can be felt makes it improbable that the condition is due to bleeding from a pregnant tube, though in some cases fresh hæmorrhage may take place into the general peritoneal cavity from rupture of the wall of an encysted collection of blood. I have known several cases of severe internal bleeding due to tubal pregnancy in which the diagnosis of perforated gastric ulcer was made, but I feel certain that in some of these the house-surgeon had intentionally made a wrong diagnosis so as to obtain dishonest possession of an interesting case. The correct diagnosis is nearly always made, and in cases where the abdomen has been opened with the idea of removing a pregnant tube which has caused severe bleeding I have never known the diagnosis to be wrong, although I have very often known the abdomen to be opened for some other condition, and tubal pregnancy with only slight intra-peritoneal bleeding found.

Simultaneous Intra- and Extra-uterine Pregnancy.

Patient, æt. 29 years, was sent up to the hospital on September 20th by her medical attendant with a diagnosis of early miscarriage, following perforation of a gastric ulcer. On September 19th she lost a large amount of blood *per vaginam* and passed a small embryo, seen by the nurse but not by the doctor. The nurse was certain that no placental tissue had been passed. She had been married five years and had two children, the younger two and a half years previously. Three days before admission, while out shopping, she had a sudden attack of violent pain all over the lower part of the abdomen, like labour pain. She vomited and felt faint. The pain went away for a short time, but returned and continued with vomiting up to the time of her admission. Catamenia: the last period ended on July 9th. After this there was no loss until the day on which the pain began, September 17th.

The patient was blanched, and was evidently in severe pain. The pulse was 130 per minute. The abdomen was distended, rigid, tender and painful. The cervix was dilated enough to admit a fingertip; no blood was coming away. Owing to the distension of the abdomen it was impossible to estimate the size of the uterus bimanually. The patient having been anæsthetised, the cervix was dilated with Hegar's dilators, and the finger passed in. The greater part of an ovum of about two months was removed with ovum forceps, and a hot intra-uterine douche was given. The abdomen was then opened, and found to contain a large quantity of blood. The left ovary and tube were found to be normal, but on the right side no ovary or tube could be found. At the right cornu of the uterus was a rupture making a rough surface about the size of a shilling, attached to which was a mass of blood-clot containing a small embryo. The cornu was excised, and the wound sewn up with catgut. A careful search failed to reveal any trace of the right tube or ovary. The round ligament on the right side was normal. There was a fold of peritoneum running from the pelvic brim to the back of the uterus rather than to the cornu, and there were some filamentous adhesions, but there were no adhesions of sufficient density to make it difficult to distinguish the nature of the structures. Projecting from the left ovary were two firm, round, yellowish bodies, each about an inch in diameter, suggestive of very small dermoids. I excised one of these, and found that it was an unusually firm corpus luteum.

For three days after the operation the temperature was 102° F. There was a good deal of dyspnœa, and there were signs of consolidation at the base of the left lung. The temperature came down by lysis and became normal on the twelfth day after the operation. The wound healed by first intention.

This case is interesting from two points of view: (1) The presence of co-existent intra- and extra-uterine pregnancy; this, though not common, is still not very rare. (2) The presence of interstitial pregnancy on the right side in the absence of a right tube.

If my opinion as to the condition of the right side of the pelvis is correct—that is, that there was no trace of ovary or tube, an opinion shared by Dr. Gordon Luker, the registrar, and by my resident accoucheur, Mr. Paget Tomlinson—this case must be looked on as one of "internal wandering" of the ovum.

Pregnancy in an Undeveloped Uterine Horn.

F. G—, æt. 29 years, was sent to me at the London Hospital on December 7th, 1911, with a diagnosis of missed labour. She had been married two years. The catamenia were regular until Christmas, 1910; after this there was amenorrhœa until October, 1911. During this period she had no vomiting and no pain. Movements were felt during May, June and July; bleeding started in the first week of October, 1911, was copious at first, and had continued as a slight flow ever since. Pain came on with the bleeding, recurred about every half hour, and lasted for about fourteen days. There was a tumour rising out of the pelvis, reaching to a point about four inches above the umbilicus on the right side, and about two inches above the umbilicus on the left side. Its upper border was irregular; otherwise it was smooth in contour, it was of almost stony hardness, was not tender, and could not be felt to contract. It had a fair range of mobility from side to side, but very little in a vertical direction. The round ligaments could not be felt. The breasts had obviously been active recently, there being much pigment present, and the secondary areolæ well marked. The cervix was high up to the left of the middle line; the body of the uterus, which seemed to be of normal size, could be felt behind and to the left of the very hard tumour. A diagnosis was made of full-time pregnancy in an undeveloped uterine horn or full-time extra-uterine pregnancy. I operated on December 11th, and found that the tumour was an undeveloped right horn of the uterus. The right ovary and tube were attached to the back of the tumour; the round ligament sprang from the middle line of the tumour at the junction of the lower and middle thirds. The uterus, of normal size, lay to the left and rather behind the tumour, being attached to it by a broad band of tissue connecting the tumour with the cervix. This connecting band was cut through, exposing a small portion of fœtal scalp. The operation was completed by section of the round ligament and separation of the tumour from the right broad ligament, the uterus and both ovaries and tubes being left behind. No opening could be seen between the undeveloped horn and the normal part of the uterus. The patient made an uninterrupted recovery. The bleeding, which had continued for two months, ceased on the fourth day after the operation. The tumour removed weighed 4 lb. 14 oz.

Acute Hydramnios.

Hydramnios—excess of liquor amnii—is seen much more commonly in the last two or three months of pregnancy than in earlier months. As a rule it causes no acute pain, although it may be responsible for a great deal of discomfort. When, however, it occurs at about the middle of pregnancy—a somewhat rare condition—it may be worthy of the term "acute hydramnios," and may cause acute symptoms, viz. severe pain and uncontrollable vomiting. In these cases it is usually found to accompany a twin pregnancy. I have seen two or three examples of this, and will read you details of one of them.

E. A—, æt. 45 years; ten children, youngest two years previously. First pregnancy twin, four miscarriages, last six years previously. Was admitted in 1904. She complained of severe abdominal pain with vomiting for one week. There had been amenorrhœa for nearly six months; the abdomen had been enlarged for four months with rapid increase in size during the last week. There was a good deal of dyspnœa. The abdomen was enormously distended, measuring 44½ in. in circumference at the level of the umbilicus. The measurement from the pubes to the ensiform cartilage was 20½ in.

These was dulness all over the abdomen except in the flanks, a cystic swelling rising up out of the pelvis filling the whole of the abdomen, reaching up to the ribs and ensiform cartilage. A fluid thrill could be obtained from side to side and from above downwards. The abdominal wall below the umbilicus was so œdematous that palpation was unsatisfactory. Fluctuation could not be obtained bimanually. There was no doubt that the patient was pregnant, as internal ballottement was obtained, but bimanual examination was very unsatisfactory on account of the œdema of the lower part of the abdominal wall, and it was impossible to say whether the large tumour filling up the abdomen was all uterus or not. The urine was free from albumen. The diagnosis lay between pregnancy with a very rapidly growing thin-walled ovarian cyst and a colossal hydramnios. Examination under an anæsthetic did not help matters, as it was still impossible to say whether the tumour was all one or not. I made a median abdominal incision just large enough to admit one finger, and found that the whole of the swelling in the abdomen was due to enlargement of the uterus. The patient was put in the lithotomy position, and the membranes were punctured with the sound. Twenty pints of liquor amnii were collected, and at least 2 or 3 pints were lost, as the water came out like a cascade. Two days later the patient was delivered of twin fœtuses of something under six months' development. I have seen two or three similar cases of acute hydramnios with twin pregnancy, and the diagnosis between hydramnios and normal pregnancy with a thin-walled ovarian cyst often presents a certain amount of difficulty, though seldom so much as in the case which I have just described.

Hydatidiform Mole.

As a rule, hydatidiform or vesicular moles do not cause severe pain, but if the mole is large and growing rapidly pain may be a marked feature of the case. In these cases there is often albuminuria also. The diagnosis of hydatidiform mole is very often not made until the mole is being passed. I think that, as a rule, the chief reason why the correct diagnosis is not made is simply that hydatidiform mole is not thought of. In cases where intra-uterine pregnancy is accompanied by pain and bleeding, or by albuminuria during the first half of pregnancy, hydatidiform mole should always be thought of, especially if there is much vomiting as well. During the last three years I have been asked to see two cases of albuminuria of pregnancy in medical wards, in which the uterus contained a hydatidiform mole. Albuminuria of pregnancy, *i.e.* albuminuria occurring for the first time during pregnancy, is so rare before the sixth month that it should arouse the suspicion that the pregnancy is abnormal. Even though the size of the uterus may suggest that the pregnancy is further advanced than would be accounted for by the period of amenorrhœa, the fact that no evidence of the presence of a fœtus can be obtained should lead to the consideration of the possibility of hydatidiform mole.

M. B—, single, æt. 21 years, complained of severe pain in the right side of the abdomen for six weeks. She had slept badly on account of the pain, and had had a good deal of vomiting. The patient looked very ill and was emaciated. She had noticed a lump in the abdomen for six weeks. The catamenia had been regular until three months previously, since when there had been amenorrhœa. An elastic swelling was found in the abdomen rising out of the pelvis and reaching up to a point above the umbilicus. Nothing could be heard over the tumour. On bimanual examination the cervix was found to be soft, and the tumour was undoubtedly the body of the uterus. A diagnosis of hydatidiform mole was made, and the mole was removed next day under an anæsthetic. I remember that in this case two or three students made a correct diagnosis independently.

Uterine Fibroids.

Acute abdominal pain due to fibroids may be caused during pregnancy either by degeneration or by impaction of the fibroids—more commonly by the former. If a uterine fibroid becomes tender and painful during pregnancy degeneration should be suspected, sometimes red degeneration, at other times some other form of degeneration. One is loth to operate on fibroids during pregnancy because the increased vascularity may make myomectomy impossible and render hysterectomy necessary, and also myomectomy *may* be followed by miscarriage or premature labour. I have only seen a few cases in which an operation on uterine fibroids has had to be performed during pregnancy solely on account of pain, and have only had to

operate myself on one case, some details of which I will read to you. In my experience rest and sedatives often enable a patient with tender painful fibroids to go on to full time. If the fibroid is painful or shows signs of degeneration after the labour, the tumour, with or without the uterus, may be removed with much less reluctance, the patient having a valuable asset in her baby. To my mind the removal of the uterus during a first pregnancy is a lamentable and grievous thing, only justified by absolute necessity, not by the fact that a uterus with fibroids in its wall is pregnant. If an operation is rendered necessary by acute pain during pregnancy, myomectomy rather than hysterectomy should be performed whenever possible.

A. R—, æt. 34 years, a widow, had one miscarriage two years ago. For five years she had noticed a small lump in the left side of the abdomen which caused no symptoms. She was admitted on September 14th, 1912. An irregular mass was found filling up the lower part of the abdomen, reaching just above the umbilicus. The cervix was soft. The mass in the abdomen could not be separated from the cervix, and a diagnosis of pregnancy with fibroids was made. She was admitted again on October 12th, 1912, as the pain was getting worse and there was great frequency of micturition every twenty minutes. The abdomen was increasing in size rapidly; six lumps of varying size could be felt in the abdomen surrounding an elastic median swelling which reached just up to the umbilicus. The median swelling seemed to be the pregnant uterus. There was definite softening of the upper part of the cervix. The pain was so severe that morphia had to be given on two occasions. She slept very badly and had to be given hypnotics. On October 15th I opened the abdomen and found that there were very numerous fibroids, varying in size from that of a pea to that of a fist. Myomectomy was out of the question, so I performed subtotal hysterectomy. On section some of the tumours were found to be undergoing cystic degeneration. The patient made an uninterrupted recovery.

Twisting of the Pedicle of an Ovarian Tumour.

This accident occurs fairly frequently during pregnancy, the tumour having to change its position to allow room for the enlarging uterus. The onset of pain in these cases is usually sudden and may be accompanied by fainting and by vomiting. If it was known before that a tumour was present it would be found that this tumour had become larger and tender, but in the majority of cases the presence of a tumour is not suspected until the twisting occurs. The abdominal wall may be so tender and so tense, intestinal distension occurring soon after the twisting, that a satisfactory examination cannot be made without an anæsthetic.

I have already referred to the diagnosis under the head of "extra-uterine pregnancy."

L. B—, æt. 28 years, was admitted in June, 1912. Six weeks before admission she had an acute attack of abdominal pain and vomited four times. She had had repeated attacks of pain up to the date of her admission. A few hours before she was admitted she had another very sharp attack of pain in the right side of the abdomen, accompanied by vomiting. She had had five children, the last two years ago, and three miscarriages. She was nine months pregnant, and was expecting her confinement any day. She looked ill and had lost a good deal of flesh. The pulse was 120 per minute. The abdomen, which was greatly distended, was asymmetrical in appearance, bulging chiefly in the right hypochondrium. There was another bulging mass on the left side where a fœtus could be plainly felt. There were evidently two distinct tumours in the abdomen. The one on the left felt like a normal uterus; fœtal parts could be felt; the fœtal heart could be heard over it. The tumour on the right side was tender and tense, and no evidence of the presence of a fœtus in it could be obtained. Below this right-sided tumour was an area of resonance. A diagnosis of pregnancy, complicated by the presence of an ovarian cyst with a twisted pedicle, was made. I opened the abdomen, letting out a good deal of blood-stained fluid, and removed a right-sided ovarian tumour, which was nearly as large as the pregnant uterus. It was a deep blue colour. There were numerous adhesions to surrounding organs and to the anterior abdominal wall, and the pedicle was tightly twisted. Labour came on seven days later and was uneventful. Both mother and child did well. I have chosen this case as being a typical example of one of the fairly common causes of acute abdominal pain during pregnancy.

The following is a good example of an obscure case of acute abdominal pain in pregnancy:

On September 11th I was asked to go down into the country to see a patient with the following history: Æt. 36 years, two children, the second two and a half years previously, now six months pregnant. On September 6th, 1912, she was sitting on the floor cutting her toe-nails. On getting up she felt a sharp pain in the right side of her abdomen. This had continued and she had been in bed since, though she could sit up without pain. There had been no vomiting. Pain was not due to contraction of the uterus, but was distinctly worse when the child kicked.

The tongue was clean, the appetite good, and the patient wanted to get up. Temperature normal, pulse 80. She had had six hours' sleep the previous night, waking twice for a few minutes. She had been rather constipated. There was a tender spot just above and internal to the right anterior superior spine, but no rigidity. The right kidney was not tender, there was no pus or albumen in the urine, and nothing abnormal could be felt abdominally or *per vaginam*. I could not make a diagnosis. Twisting of an ovarian tumour, appendicitis, pyelonephritis and degeneration of a uterine fibroid all seemed to be excluded. I thought that nothing serious could be the matter, and ordered rest, mustard leaf, and aperients.

On the 13th the abdomen became somewhat distended, and on the next day was greatly distended in spite of enemata. The patient vomited only after castor oil. I saw her at 4 p.m. The pain was much more severe. The abdomen was enormously distended—a binder which had overlapped widely could not be made to meet. There was visible peristalsis. The patient was much distressed by the distension and looked ill. Examination revealed nothing beyond the distension except that the tender spot was still present as before. It was evident that the abdomen must be opened. I thought that I must have missed a small ovarian tumour which had become twisted, but as the diagnosis was still obscure I got my colleague, Mr. James Sherren, to go down with me in the evening.

We operated at 9.30 p.m. On opening the abdomen and exploring the right iliac fossa a small purple body came into view, which was at first thought to be a gangrenous appendix, but was found to be attached to the fimbriated extremity of the right Fallopian tube. It was about two inches long, three quarters of an inch thick, and was constricted in the middle of its length. It had the appearance of being some soft structure which had become deeply congested and then become practically solid as the result of thrombosis. The tube, ovary and appendix were healthy and normal. The small purple body was removed after its attachment to the fimbriated extremity of the Fallopian tube had been ligatured with catgut. Nothing else abnormal was found in the abdomen. The abdomen was closed and the patient put back to bed. A sixtieth of a grain of strychnine was given four-hourly. The distension, which had seemed to be rather less when the patient was returned to bed, became more marked during the course of the next day. She was given a turpentine enema at 10 a.m., which caused the evacuation of a large amount of flatus, and another enema at 5 o'clock brought away a small quantity. I saw her at 8 p.m. She had been sick five times during the day; the pulse was about 116. She was rather distressed by the distension, and complained of some pain in the right side of her chest. There was no pain in the right iliac fossa. She was given 100th of a grain of eserine and some hot brandy and water, was propped up in a sitting position, and was given a two-pint turpentine enema through a long rectal tube. A large amount of flatus was passed and the patient felt much relieved. After this progress was satisfactory, although there was a large amount of distension for several days.

Under the microscope the piece of tissue was found to consist of a thin-walled cyst with some lymph on its surface, the interior filled up with blood-clot. It was apparently a twisted hydatid of Morgagni, which could not have been larger than a small grape before torsion occurred. The pregnancy terminated in an uneventful labour. The case seemed to me to be worth recording, as the condition was a very unusual one and a correct diagnosis almost impossible.

Intestinal Distension.

Cases of *intestinal distension* during the later months of pregnancy are rather alarming, as the patient soon becomes distressed by the extra tension in an abdomen which is already full, and looks worse, if I may be allowed to put it that way, than she ought to. A short time ago I was called down into the country one evening to see a patient and told to come prepared to operate. The patient was æt. 23 years, eight months advanced in her first pregnancy. Two days before I saw her she was perfectly well until after dinner, when she was seized with pain and discomfort in the abdomen. These passed off after a few hours, and she was up

and about the next day until the evening, when the pain returned. She had pain all night of sufficient severity for the doctor to stay with her or in a room near by all night, and to send for a colleague to see her with him. She vomited once; a turpentine enema brought away some scybala. She said that during her pregnancy the bowels had been opened regularly, and that she had taken aperients when necessary. She was a very nervous girl, asthmatic, and a good deal alarmed about her condition. The pulse was about 90 per minute; temperature normal; tongue furred. She complained of severe pain in the right side of the abdomen extending into the flank, with a good deal of tenderness. The ascending colon was distended and very tender on slight pressure, but firm pressure caused her a certain amount of relief. There was no real rigidity; there was pain on taking a deep breath, and pain on any movement of the body. Otherwise abdominal and pelvic examination revealed nothing abnormal. After discussing and excluding appendicitis, pyelonephritis, twisting of an ovarian tumour, and diaphragmatic pleurisy, I came to the conclusion that her symptoms were due entirely to constipation and distension, and ordered an ounce of castor oil with 10 min. of tincture of opium, to be followed in the morning by another turpentine enema, and fortunately warned her mother that labour might come on as a result of the disturbance. Labour came on next day, and all went well, with no return of the pain. For the next few days enemata brought away a large number of foul-smelling pale-coloured scybala. It must be remembered that a statement made by the patient that the bowels are opened regularly is not inconsistent with the presence of chronic constipation. The bowels may be opened daily, but each evacuation may be a day or two late.

Pneumonia and Pleurisy.

Fortunately pneumonia and pleurisy are not very often seen in pregnant women. It is possible that a woman during pregnancy may have a certain amount of extra immunity. I can only remember one case of pneumonia, post-influenzal, complicating pregnancy in a case of my own. There was so much distress due to the impeded movements of the diaphragm that we had to consider the question of terminating the pregnancy. While we were considering—being much averse from doing it if we could possibly help it, the pregnancy of about thirty weeks being the first in a woman, æt. 43 years—premature labour came on spontaneously. I can imagine that diaphragmatic pleurisy in the later months might cause great difficulty in diagnosis before physical signs developed. I have had to consider it once or twice, but have not so far come across a case in which it was present.

September 8th, 1913.

A CLINICAL DEMONSTRATION OF FIBROSIS OF THE LUNG AND BRONCHIECTASIS IN CHILDREN.

Delivered at St. Bartholomew's Hospital.

By H. MORLEY FLETCHER, M.D., F.R.C.P.,
Physician to the Hospital; Physician-in-Charge of the Children's Department; Physician to the East London Hospital for Children.

Gentlemen,—I have chosen for this morning's demonstration the subject of fibrosis of the lung and bronchiectasis, and I have done so for three reasons. In the first place because, in my opinion, these conditions in children are much commoner than you would be led to believe from a perusal of the text-books on the subject; secondly, they are apt to be overlooked or to be mistaken for other pulmonary conditions; and thirdly, and more important still, because they are not infrequently mistaken for cases of chronic pulmonary tuberculosis. It is for those reasons that I have thought it well to bring these cases before you to-day. You must bear in mind, however, that pulmonary tuberculosis, of the ordinary chronic form, such as one sees in adults, is quite rare in children; it is very seldom that one comes across cases of chronic pulmonary tuberculosis of the adult type in children under ten or twelve years. Pulmonary tuberculosis in children is common enough, but it must be remembered that the forms met with differ from the fibro-caseous variety of the adult. In children acute miliary tuberculosis and tuberculous broncho-pneumonia make up the great majority of cases of pulmonary tuberculosis.

I shall first say a few words about bronchiectasis, and then refer to the subject of fibrosis of the lung, and I shall show you cases to illustrate these conditions.

The production of bronchiectasis or dilatation of bronchi depends upon three factors: first, an increase of intra-bronchial pressure. Anything which raises the pressure in the bronchi will tend to distend them. For that reason we find that a chronic condition of cough in children, such as whooping-cough, may give rise to bronchiectasis.

Secondly, any condition which produces softening of the walls of the bronchi will tend to produce dilatation: I refer to inflammatory conditions such as broncho-pneumonia, bronchitis, etc. Thirdly, there is a factor less common in children than the foregoing, namely, traction on the walls of the bronchus. This cause of bronchiectasis is found in fibrotic conditions of the lung in which the walls of the bronchi are dragged upon by the contracting fibrous tissue.

It will be obvious to you that a child's bronchus is more liable to dilatation than is the bronchus of an adult. In the child the wall is thinner and more readily affected by inflammatory conditions, and the dilating action of cough is more intensely felt than in the adult.

Bronchiectasis in infancy and early childhood may be either acute or chronic. The *acute* form is most frequently met with in infants in the course of acute bronchitis or broncho-pneumonia. The condition may be unsuspected during life, and post-mortem the bronchi are found to be obviously dilated and inflamed. It is often possible to recognise during life the existence of this form of bronchiectasis by the presence of coarse *râles* over the bases of the lungs behind which persist for some length of time after the acute stage has passed off. The dilatation in these cases is due to inflammatory softening of the bronchial walls and to increased intra-bronchial pressure, caused by coughing. The condition in the great majority of cases is not a persistent one, the inflammation subsides, and as the growth of the lungs proceeds the bronchial tubes may assume their relatively normal size.

Bronchiectasis of a more chronic type is not uncommonly met with after whooping-cough, and it is remarkable how often this condition is overlooked. In many cases of protracted whooping-cough, both in small infants and in other children, it will be found, even months after the ordinary manifestations of whooping-cough have ceased, that there are signs of bronchiectasis at the bases indicated by coarse bubbling *râles* and less commonly by areas of abnormal breath-sounds, *i. e.* distant bronchial or amphoric breathing, with bronchophony. In such cases muco-purulent expectoration persists long after the paroxysmal cough has ceased. The cough and expectoration may last for a year or even longer after the original attack of pertussis has subsided and then gradually diminish and the signs in the chest clear up. A few less fortunate cases may remain permanently affected by chronic bronchiectasis. I have three or four cases to show you to-day illustrating the fact that long after the subsidence of the original disease such children may continue to bring up large quantities of phlegm and to exhibit the signs of bronchiectasis.

In favourable cases the catarrhal condition clears up gradually and the child regains strength. The development of the lung proceeds, and the previously dilated tubes appear to become of normal size relative to the lung, so that in course of time all signs of bronchiectasis disappear. But on the other hand, a certain number of these cases do not clear up, but remain permanently affected with bronchial catarrh and bronchiectasis.

The signs of bronchiectasis in children vary with the degree of dilatation of the tubes which has taken place. In the slighter and more acute forms they consist of more or less persistent *râles* at the bases of the lungs, best heard at the back, and usually accompanied by a varying amount of a general bronchial catarrh. If accompanied by unresolved broncho-pneumonia and collapse, there may be impairment or dulness to percussion, and bronchial breathing. If the bronchiectatic cavities are large, amphoric breathing may be heard over one or more small areas at the bases behind.

As regards the *symptoms* of bronchiectasis there is one point on which I wish to lay special emphasis, and that is the occurrence of expectoration. The existence of dilatation of the bronchi should be always suspected when a history is obtained that a child coughs up sputum. There are practically only two conditions in which a child brings up sputum with cough; one is whooping-cough and the other bronchiectasis. In nearly every case I have to show you this morning what directed the mother's attention to the condition of the child was the frequent expectoration after cough.

Dilatation of the bronchi is usually most marked in cases with fibrosis of the lung, to which I shall refer later, and in these the quantity of sputum is more abundant than in those with simple dilatation without fibrosis.

I shall now show you a few cases illustrating the condition of bronchiectasis.

This little girl, æt. 6 years, had whooping-cough in November, 1911, ten months ago, and since that

date has not been free from cough. She has frequent and violent attacks of coughing in which she brings up copious purulent expectoration, occasionally with slight hæmoptysis. She has been under observation for some months and usually presents the signs you will now find, namely normal resonance to percussion over the lungs with coarse, almost bubbling *râles* at the bases behind. When requested to do so she will cough up large plugs of sputum, and you will observe that after coughing most of the *râles* will disappear. Her condition, both general and local, is gradually improving.

The next patient, a girl, æt. 5 years, had whooping-cough eleven months ago, and since then she has had a persistent cough with which she brings up large quantities of purulent sputum. The signs in the chest closely resemble those in the first patient.

Next is a boy, æt. 10 years, who had measles and "pleurisy" at six years and had had chronic cough and expectoration ever since. On rising in the morning he brings up green phlegm occasionally streaked with blood. He is a delicate-looking boy and his state of general nutrition is poor. The signs usually present in his lungs consist of coarse *râles* limited to the bases clearing up almost entirely after coughing.

These cases illustrate the frequent occurrence of simple dilatation of the bronchi in children as the result of inflammatory conditions and of chronic cough, especially pertussis. I wish to emphasise that some degree of bronchiectasis may be observed more often than is generally recognised during or after an attack of broncho-pneumonia or whooping-cough if attention be directed to the physical signs.

I now pass to the condition of bronchiectasis associated with fibrosis of the lung. Fibrosis of the lung in children, as in adults, may be either localised or diffuse—that is to say, fibrosis may be present either as a localised patch, or as a large area involving the greater part of a lobe or even the whole lung. But in children it is much less common to find localised fibrosis than in adults, in fact it is a rare occurrence, and is not of great clinical importance, so that I do not propose to deal with this. Diffuse fibrosis, on the other hand, is fairly common, and may result from the following causes. It may be due to non-expansion of a portion of the lung, as in fœtal or congenital atelectasis, the unexpanded part ultimately becoming fibrous. Conditions leading to pulmonary collapse may cause fibrosis, such as pleural effusions and obstruction of a bronchial tube or of the smaller bronchi. Catarrhal states of the lungs are very apt to produce collapse, especially in young and feeble children; the tubes are narrow and therefore easily obstructed, while the weak respiratory muscles are unable to produce a cough sufficiently strong to dislodge the mucus in the tubes and to expand the lungs.

Inflammatory conditions such as broncho- and lobar pneumonia, especially the former, are the most important factors in producing fibrosis. In broncho-pneumonia collapse is commonly present owing to the plugging of the tubes with secretion, while at the same time inflammatory exudation takes place into the alveoli, the peribronchial tissue and the interstitial tissue of the lung. Fibrosis may result from the organisation of the inflammatory products, and the whole of one lobe may ultimately become densely fibrotic, so that little or none of the normal spongy tissue can be detected. The lower lobes are those most commonly affected, probably owing to their greatly liability to collapse. The walls of the bronchial tubes, already softened by the inflammatory process, are further subjected to the distending influence of the chronic cough which is almost always present in these cases, and the increased intra-bronchial pressure caused by coughing leads to the production of bronchiectasis in the affected part of the lung. As fibrosis proceeds the bronchi may also undergo dilatation as the result of traction on their walls by the newly formed and contracting peribronchial fibrous tissue. In the majority of cases of fibrosis of the lungs in children some degree of bronchiectasis is present. Pleurisy, especially the forms producing considerable thickening of the pleura, may lead to fibrosis of the lung; the formation of fibrous tissue appears in such cases to extend from without inwards into the interlobular tissue of the lung, and the whole lobe may become involved, but this is much less common in children than in adults.

When the lobe of a lung becomes fibrotic it contracts and occupies less space than it did formerly. To compensate for this that side of the chest may fall in, or the mediastinum may be dragged over to the affected side, or the diaphragm may be drawn up. In some cases we find these three compensatory displacements have taken place, but it is noteworthy how frequently we meet with examples of fibrosis of the lung in children

with little obvious asymmetry of the thorax, and I believe that this depends on the absence of pleural adhesions in such cases. There is usually some degree of scoliosis present in cases with contraction of one side of the chest.

Fibrosis of the lung affects the lower lobe of the left much more commonly than that of the right lung, and I do not think sufficient attention has been paid to this point. I cannot give off-hand figures to show the relative frequency on the two sides, but among the many cases I have had under observation those in which the left side was involved are far the more numerous. I believe that this is probably due to the fact that the left lower lobe is more liable to undergo collapse than is the right lung, owing to the position of the heart. Pressure exerted by the heart may predispose to collapse, or may be a determining factor in preventing or retarding re-expansion of the left lung.

In a considerable proportion of cases of fibrosis of the lung with bronchiectasis general nutrition is poor and bodily growth may be retarded. The face is usually pale and sometimes is inclined to be puffy; the cheeks are congested. I have noticed in some advanced cases a curious fulness in the parotid region, of the nature of which I am ignorant. The fingers are often clubbed, but not always; some of the cases here to-day exhibit this, others do not do so.

The expectoration is usually a greenish muco-purulent fluid. It is coughed up in the form of large plugs or may come up in quantity as a copious stream, not uncommonly pouring from the nose as well as the mouth, and so giving rise to the mistaken diagnosis of vomiting. The sputum is often offensive, but the degree of fœtor varies in individual cases. In winter the quantity of expectoration is usually greater owing to increased bronchial catarrh, and the offensive smell may return; streaks of blood are frequently present and sometime severe hæmoptysis occurs, which may lead to a wrong diagnosis of pulmonary tuberculosis.

Occasionally cases are met with in children in which the sputum is persistently swallowed in spite of its large quantity, and I have known cases in which it has been necessary to administer an emetic or to wash out the stomach to get rid of the infected material.

I shall now show you cases to illustrate these points and to demonstrate the physical signs.

This boy of six has fibrosis of the left lung with bronchiectasis. He has had cough from infancy, which has become much worse during the last three years, and for the past twelve months he is said to have vomited with the cough. At times he becomes cyanosed, especially after coughing. The thorax shows slight asymmetry: the left side is slightly contracted, especially when viewed posteriorly, and does not expand as well as the right side. The apex-beat of the heart is displaced towards the left axilla. The percussion note in the left axilla and over the base of the left lung behind is greatly impaired, and below the angle of the scapula there is loud bronchial breathing almost amphoric in character with bronchophony and whispering pectoriloquy—at times bubbling *râles* are heard over this area. These signs are due to fibrosis of the left lower lobe with dilatation of the bronchi. There is no evidence of pulmonary tuberculosis, though I may say that he was sent from school as he was supposed to be suffering from that disease.

Here is another case very similar to the preceding, but with signs even more marked—namely, displacement of the heart to the left and great dulness over the left base behind and in the axilla. This case illustrates an interesting clinical point. At present the bronchiectatic cavities at the base are filled with secretion and the breath-sounds behind are heard very faintly, if at all. I will now tell the boy to cough, and will invert him over this chair to aid him to empty his bronchial tubes, and you see that he brings up a large quantity of thin green purulent sputum which is not fœtid. On listening to the back once more we now hear loud amphoric breathing with some coarse *râles*; the air is able to pass through the dilated tubes previously blocked by secretion. This variation in the physical signs is not uncommon in these cases.

I have mentioned the displacement of the heart and mediastinum towards the affected side. This is due, in part at least, to the increase in size of the other lung, the result of compensatory hypertrophy. The normal resonance of the sound lung is found to be considerably increased and to extend well over to the other side. I have brought down from the museum a preparation of the thoracic viscera which illustrates exceedingly well this compensatory hypertrophy.

The next case is a boy, æt. 6 years, who suffers from cough dating from an attack of pneumonia and bronchitis lasting six weeks in his second year. He has a good deal of expectoration, and sometimes discharges as much as a cupful of offensive sputum, which is occasionally streaked with blood. His fingers are clubbed. There is no obvious recession of the chest, but a cyrtometer tracing shows that there is a slight difference between the two sides. The apex-beat is 1½ in. outside the nipple line. The signs at the left base are the same as in the preceding cases. Like the second case I have just shown you, this boy can bring up large quantities of expectoration when told to do so, and he exhibits the same resulting variation of signs at the base. At present the sputum is odourless, but in winter it is usually very fœtid.

Other cases of pulmonary fibrosis were also exhibited, including one involving the right lower lobe.

Prognosis.—I shall now deal with the prognosis. This largely depends on whether there is only fibrosis of the lung, or whether it is associated with bronchiectasis. If there is fibrosis without bronchiectasis the prognosis is better than if bronchiectasis is present. The fact that the patient has a limited functionless area of lung does not necessarily mean that it is dangerous to life, because other parts of the lungs readily take on its functions by undergoing compensatory hypertrophy. If there is no intercurrent or secondary disease such as pneumonia, pulmonary tuberculosis, chronic bronchitis, etc., the patient may get along very fairly well. Unfortunately many of these cases with fibrosis have dilated bronchi, and the prognosis in these must depend on the size, situation and number of the cavities, and also on the degree of infection which has taken place within them.

Bronchiectasis of the chronic variety must be looked upon as an added danger to life, though the acute form already mentioned is not so serious. The dangers of bronchiectasis are infection, chronic catarrh and hæmoptysis.

The bronchial tubes and the cavities become infected by a variety of organisms, thus causing an abundant and often fœtid secretion, which often gives rise to progressive anæmia and asthenia, so that the child readily succumbs to some intercurrent disease. Or the infection may give rise to a general septicæmia.

Another well-recognised termination in children as well as adults may be brought about by the development of a cerebral abscess. Not long ago a boy under my care suffering from pulmonary fibrosis and bronchiectasis came up complaining of headache with occasional vomiting and some weakness of the arm, and died a few days later with a solitary abscess in the brain which trephining and exploration failed to relieve.

Lardaceous disease may result from the constant purulent discharge, but I have not seen an instance of this that I can recollect.

Hæmoptysis is common and is usually slight, the blood oozing from the ulcerated bronchial mucous membrane. Occasionally it is severe and may be fatal. The occurrence of hæmoptysis is an event which may lead to the erroneous diagnosis of pulmonary tuberculosis.

Blocking of the outlet of a cavity filled with infected secretion may give rise to a condition closely resembling pulmonary abscess. The contents may burst into the pleural cavity if this is not already obliterated by adhesions and thus cause empyema.

A small bronchiectatic cavity situated in the upper part of the lung is a more favourable condition than one situated at the base, owing to the greater facility it will have for emptying itself.

I have told you that one of the dangers in fibrosis of the lung, either with or without bronchiectasis, may be the supervention of tuberculosis, and I have here a case to illustrate this point. This girl, æt. 12 years, has had cough with copious and often fœtid expectoration since she was five. The sputum has often been blood-stained. She has fibrosis of the right lower lobe. The apex-beat of the heart is just internal to the right nipple line. It is interesting to note that there is no obvious asymmetry of the thorax. The signs over the right lower lobe are exactly like those I have already described as resulting from fibrosis and bronchiectasis. A few weeks ago she became much worse and developed pyrexia, and we found tubercle bacilli in the sputum, and she is now going rapidly down-hill. I have seen similar cases, and the condition post-mortem is generally one of recent tuberculous broncho-pneumonia with tuberculous bronchial glands without evidence of chronic pulmonary tuberculosis.

Treatment.—I fear that the time left at my dis-

posal will not permit me to deal with the treatment of bronchiectasis as fully as I should like. Although unfortunately in many cases the condition must be regarded as incurable, yet much may be done to alleviate it and to make life more pleasant for the patient. In some cases great improvement may be brought about.

Treatment should be directed (1) to improve the state of the general health by good feeding, fresh air and sunshine, and by the administration of tonics. (2) To reduce the tendency to bronchial catarrh by suitable climate: preferably the child should reside at the seaside, and, if possible, winter abroad. (3) To the regular emptying of the dilated tubes of the accumulated secretion as completely as possible. This is always difficult, but much may be done with perseverance. With small children this may be facilitated by holding the patient inverted with the head down and the feet raised, at the same time encouraging him to cough. The fluid gravitates downwards, and is more readily expelled in this position. Older children may be instructed to lie over the side of the bed or a sofa with the head touching the floor and to cough in this position until expectoration ceases. It is important that the tubes should be thoroughly cleared out first thing on rising in the morning and at regular intervals during the day and before going to bed. Unless these or similar measures are taken a large amount of the septic secretion remains in the lung and tends to aggravate the condition, or is swallowed as sputum and may give rise to disturbance of the alimentary system. It is not difficult with perseverance to train children affected with this disease to expectorate the sputum instead of swallowing it. (4) To render the cavities as aseptic as possible. I have seen little good in this respect result from the internal administration of drugs except in so far as they aid in raising the general resistance to infection. Inhalation methods are much more efficacious, especially the creosote treatment introduced by Dr. Arnold Chaplin. In this the patient is placed in a small chamber which is filled with creosote vapour. This induces coughing and a large amount of secretion is brought up, and the bronchial tubes are exposed to the deodorising and disinfecting action of the creosote. Repeated treatment often leads to a great diminution in the amount of secretion, to loss of fœtor, a cessation of pyrexia and a gain in weight. Good results are also obtained by continuous inhalation of creosote and other drugs through a respirator covering the nose and mouth worn both night and day.

Vaccines have been employed in the treatment of bronchiectasis, but in my experience have been of little benefit.

It must be obvious to you all that none of the lines of treatment I have referred to can be regarded as radical; they do not do more than lessen the evils caused by the presence of these cavities; they are not curative. Surgical measures have been employed, but so far with little success. Opening the chest and draining the cavities would seem a simple enough operation, but the cavities are multiple in most cases, and you will see from the specimens I have here, that the affected lower lobe presents a honeycomb appearance and could not be properly drained. Resection of a portion of the chest-wall to promote complete collapse of the lobe offers a better chance of success, though complete removal of the involved and useless portion of the lung, when surgical methods will safely permit of such procedure, would seem to be the most rational treatment in a severe case.

September 8th, 1913.

A Method of Determining the Extent of the Circulation in Cases of Gangrene.—Sandrock discovered this very simple method accidentally. He was amputating the leg. He noticed that the thigh had turned red under the rubbing, but only down to a certain point on the leg, where the reactive hyperæmia stopped abruptly, and below, although the parts had been rubbed equally vigorously, the leg was still absolutely pale. Other tests were applied, all showing the same limit of the blood-supply at this circular boundary. He has applied the test in various cases of diabetic or vascular gangrene, and always found that the diffuse redness of the well-nourished part of the limb stopped short at the limit of the defectively nourished part. When he has amputated at this line he has never regretted it. He controls the test by rubbing the sound leg at the same time. The difference is marked.—*Journ. A.M.A.*, vol. lxi, p. 446.

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REMARKS ON TUBERCULOSIS OF THE CERVICAL GLANDS.*

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THE preventive measures for tuberculosis of the lungs apply equally well for tuberculosis elsewhere, and consist in preventing the bacillus from gaining access to the body. In tuberculosis of the glands the infection is, of course, always secondary, and we can do much, by removing a primary focus, to prevent the glandular infection becoming extensive and widespread. It is so well known that the body is able to deal successfully with a limited amount of this disease that after the removal of the source of bacilli the tendency is towards natural cure. In this particular form of the disease preventive treatment is most important. It saves the long duration of illness and consequent expense. It eliminates deformity and saves surgical time, for one feels each time it becomes necessary to remove diseased lymphatic glands that one might have been spared the trouble, and milder measures would have sufficed, had the case been efficiently treated earlier. Although a dissection of the neck glands forms an excellent surgical exercise, one would much prefer being spared the necessity of performing it, and I trust that before very long the operation will become almost obsolete.

To appreciate and understand enlargement of the neck glands it is essential to know their situation, grouping, the areas from which they receive lymph, and the further groups into which they

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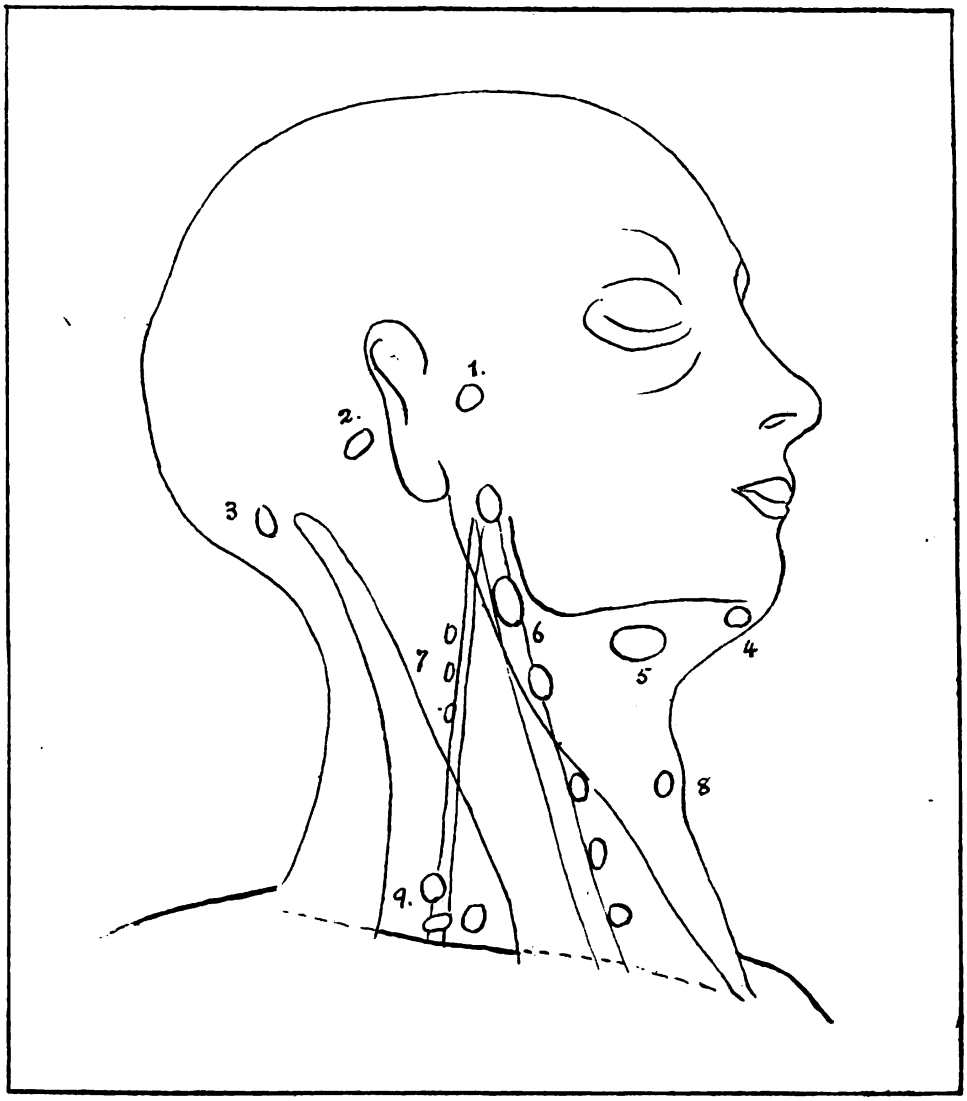

FIG. 1.—Diagram to illustrate the groups of cervical lymphatic glands. 1. Parotid group. 2. Mastoid group. 3. Occipital group. 4. Sub-mental group. 5. Sub-maxillary. 6. Deep cervical group. 7. Superficial cervical group. 8. Pre-tracheal group. 9. Supra-clavicular group.

themselves drain (Fig. 1). There are *three main groups* in the cervical region, each having several sub-groups. These are—

I. The *peri-cervical* group, forming a ring round the neck as follows:

(1) Sub-occipital.
(2) Mastoid.
(3) Parotid.
(4) Sub-maxillary.
(5) Sub-mental.

II. The *descending cervical* glands, receiving lymph from the pharynx and from the above groups of glands. They consist of two sets:

(1) Those under the sterno-mastoid muscle and arranged around the internal jugular vein.

(2) Those situated in the supra-clavicular fossa.

III. Certain *accessory* groups:

(1) Situate along the external jugular vein.

(2) *The superficial anterior cervical*, along the course of the anterior jugular vein.

(3) *The deep anterior cervical*, situated in front of the trachea, larynx and thyroid gland.

(4) *Recurrent glands*, in the interval between the trachea and œsophagus in relation to the recurrent laryngeal nerve.

These groups each drain definite areas, and it is easily remembered that the lymphatics correspond closely with the veins and drain almost identical areas.

Thus the lymph coming from the side of the head passes first to the parotid or mastoid glands; from here it passes to the deep cervical chain, along the course of the internal jugular vein, and may still pass through some glands in the supra-clavicular triangle before it enters the thoracic duct.

With this knowledge it is possible when any set of glands becomes infected to examine at once the region from which it drains lymph.

It is essential to realise that glandular disease is almost always secondary, and therefore some primary focus must be sought as the origin of the infection. Primary disease of the lymphatic glands is rare; lymphadenoma and leukæmia are the commonest ones. The focus of infection may be *obvious*; it, on the other hand, may be *small*, or *hidden*, or even *healed* by the time the patient presents himself for treatment.

The common sources of infection are:

(1) Pediculosis capitis.

(2) Suppuration of the middle ear, due most often in young children to tuberculosis of the petrous bone, of which I have had four cases this year. The onset is insidious, and facial paralysis or enlarged glands may be the earliest signs. A discharge from the ear only ensues when secondary infection has occurred.

(3) *Carious teeth* are a frequent source of infection, and tubercle bacilli have been demonstrated in their cavities.

(4) *Tuberculosis cutis*, primarily involving the skin. I have at present a case in my ward which has caused extensive tuberculosis of the cervical glands.

(5) The supra-clavicular glands may become infected from those in the *mediastinum*, and this sequence should always be suspected if the glands above the clavicle are the earliest to enlarge.

(6) The glands may be enlarged in *general tuberculosis*, which is responsible for so many deaths in children.

(7) Lastly, and most important of all, is infection from *tonsils* and *adenoids*. These two sources account for 80 per cent. of tuberculous cervical glands, and I cannot emphasise too much their importance. The tubercle bacilli gain access to the throat and naso-pharynx most frequently from the cow's milk. The admirable researches carried out by Mr. Stiles and his assistants in Edinburgh have amply proved this fact, for not only have they shown that the bacillus is nearly always of the bovine type, but they have traced the source of the milk supply to the actual tuberculous cattle. The Eustachian tube probably becomes infected in the same way, and leads to tuberculous disease of the tympanum and mastoid antrum. In other cases the source of infection must be different, because I have at present a case of tuberculous mastoid and cervical glands in a patient, æt. 3 months. It is still being breast fed, and the mother shows no evidence of tubercle. In such cases the bacilli probably gain access to the pharynx by being inspired. Tubercle bacilli have been repeatedly demonstrated in adenoids, which occur much earlier in life than is commonly supposed and form a frequent source of infection.

I am convinced, however, that the tonsil is the chief offender, and in practically every case of enlarged tonsils the glands at the angle of the jaw will be found palpable and diseased.

Whether such glands are tuberculous from the beginning it is impossible to say positively. When they reach a definite size and are of a chronic nature they can be assumed to be tuberculous. It is surprising to find in the post-mortem room that such small glands show definite areas of caseation, although clinically the great majority disappear when suitable treatment is adopted.

The enlarged tonsil is generally readily recognised as a possible focus for infection, and it is frequently tuberculous. When so, it appears slightly yellowish or pale in colour and is unduly friable, but the microscope is needed to give a definite opinion. Tuberculous ulceration is rare. It is important to realise that the enlarged tonsil may be embedded and not project beyond the faucial pillar, and this may be overlooked unless one is familiar with the condition (1). It is more important to realise that the tonsil may be quite small and yet tuberculous, and that when so the diseased portion is nearly always situated in its deepest part, close to the capsule. Such tonsils

FIG. 2.—Posterior aspect of a tonsil. Removed from a patient with tuberculous glands. The light spots are caseous areas under the capsule.

can be definitely shown to be diseased only on removal, but if there were enlarged glands in the tonsillar region, just below and behind the angle of the jaw, then this is sufficient reason for tonsillectomy. To remove the whole focus of infection the tonsil must be enucleated. This enucleation can be readily carried out by means of the guillotine (2). On numerous occasions last year I enucleated the stumps of tonsils for tuberculous glands, where tonsillotomy had been performed some time previously. In each case I was able to demonstrate a caseous focus in the part removed. There may be a single caseous focus, as in some of the specimens shown, or each lacuna may contain a small caseous nodule (Fig. 2). These foci give no *local* evidence of their presence, and sore throats are entirely absent (3).

When the bacilli pass from such a focus into the gland a *miliary tubercle* is formed. A gland occupied by a number of these becomes enlarged, though its appearance is little changed, and the condition is known as fleshy enlargement (Fig. 3). In some cases a large mass of glands may be in this condition and no caseous areas are seen. There seems to be very little tendency to break down, and such cases are confused with lymphadenoma. So far as I have been able to ascertain, this absence of caseation depends rather on the patient than on the type of bacillus, for it has not been shown to be due specially to the human or bovine type. However, this variety of enlargement is rare, and the microscope readily reveals its tuberculous nature. In most cases quite small glands, not much larger than peas, will be found to have passed on to caseation.

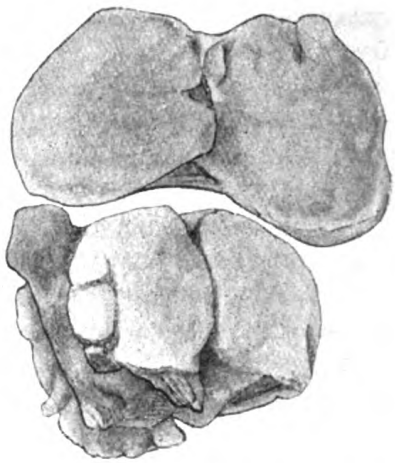

FIG. 3.—Tuberculous glands in stage of fleshy enlargement. No sign of caseation. All the glands were of this type.

If the primary focus becomes cured, or is removed at this stage, the glands will almost certainly undergo fibrosis, and a natural cure results. More frequently *caseation* follows: starting at numerous points, it finally merges into one or

FIG. 4.—Small gland showing multiple foci of caseation.

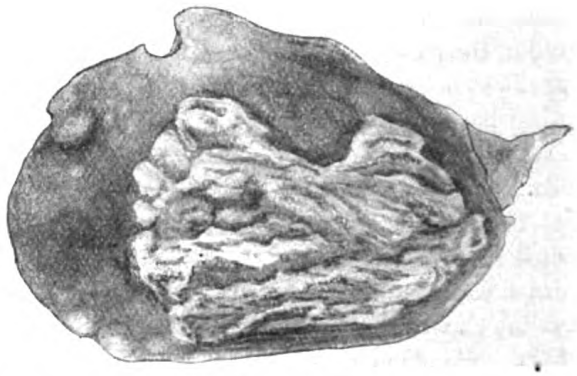

FIG. 5.—Large gland showing caseous focus From the same patient as the above. Fig. 2 is the tonsil from this case.

more masses which occupy a large part of the gland (Figs. 4 and 5). At this stage calcification may occur, and though the disease becomes temporarily quiescent, it may again become active some years after. The calcareous mass illustrated (Fig. 6) was found projecting through a hole in the deep cervical fascia, at the bottom of a large tuberculous abscess in a patient, æt. 18 years, who during childhood had suffered from enlarged glands. More recently I have seen three cases of chronic abscess occurring in the neck, just below and behind the angle of the jaw in persons between twenty-five and forty years of age. The throat gave no clue to their origin. In one case the abscess was drained, only to return some months later, when total removal of the gland was necessary. In another case, after evacuation of the abscess, two small holes were found in the deep fascia leading into caseous and calcareous glands, which were removed with the spoon. These glands had been infected during childhood, but had remained quiescent for a number of years, then had again become active and given rise to a so-called residual abscess. When caseation has been established, the gland, though, it may shrink considerably, remains permanently enlarged for many years. Caseation may pass on to softening, and then invasion of the neighbouring structures takes place. The gland infects its fellows, and one generally finds one large gland and numerous smaller ones near or adherent exhibiting an earlier stage of the disease. The gland also becomes adherent to the *sterno-mastoid* muscle, and this may become infiltrated. The *subcutaneous tissue* becomes involved and an abscess forms in it. It is at this stage that cases are frequently sent for treatment, for a prominent swelling appears and causes attention. This subcutaneous abscess may burst and leave a sinus which persists so long as the deep gland contains caseous material. Frequently this abscess is scraped out, under the impression that the whole of the disease is being removed, but the gland in reality is not touched, as it is situated under the deep fascia and communicates with the abscess by a small opening, which is overlooked (4) (Figs. 7 and 8). The *internal jugular* vein rarely escapes, and dense adhesions may form, so that in the removal of glands it may be necessary to remove a piece or almost the whole length of that structure (Fig. 9). Should ulceration

FIG. 6.—Calcareous masses (natural size) found at the bottom of a tuberculous abscess in the neck.

FIG. 7.—Tuberculous mass removed from the neck. A. Subcutaneous abscess. B. Tuberculous glands. The constriction between the two parts corresponds to the situation of the deep fascia.

and perforation occur into the vein, a general miliary tuberculosis or deposits in the lung results.

After the healing of such a sinus or tuberculous abscess an unsightly scar results from puckering of the skin or consequent tuberculosis cutis.

Tuberculous glands are readily recognised and their signs are well known. The point of most

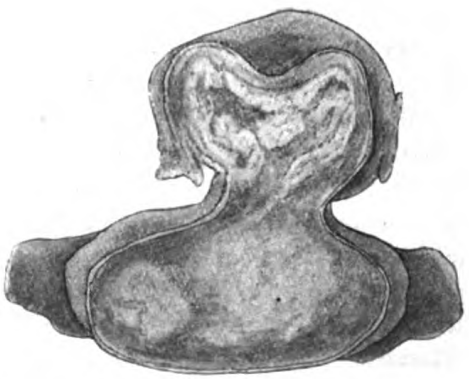

FIG. 8.—The above specimen on section. The caseous material is seen passing from the gland and infecting the subcutaneous tissue.

FIG. 9.—Mass of tuberculous glands, from a child æt. 3 years. A. The internal jugular vein, which was surrounded and infiltrated and had to be removed. The tonsil on the corresponding side was found to be tuberculous.

importance is to realise that they are secondary manifestations of tubercle elsewhere, and this primary focus must be sought. It is also essential to recognise the stage that they have reached as the treatment varies with this condition. Small hard glands will probably subside, but when once as large as a filbert nut or walnut, they will contain large caseous areas and probably demand removal. Softening generally means a subcutaneous abscess, whilst their adherence to the skin or deeper structures should be determined. In the early stages they are frequently single, but at a later period others become enlarged and may form a large mass or chain. In all these cases due allowance must be made for small glands which are impalpable, but will almost certainly be present. The glands are frequently enlarged on both sides of the neck, although one side is often greatly in advance of the other.

The differential diagnosis is rarely difficult if due allowance is made for the relative frequency of the diseases. Tuberculous glands occur every day in hospital practice, whilst primary diseases of a different nature are rare.

Dermoid cysts of the neck, arising from the branchial clefts, are rare; no case occurred at the Children's Hospital during 1912. They are always single, and situated chiefly in front of the sterno-mastoid muscle.

Lymphadenoma is also rare, no case occurring at the same hospital last year. I know of no physical sign except softening which will definitely decide between these conditions, and this is not infallible Enlargement of the spleen at a later stage may prove helpful. Microscopic examination of a gland is the only crucial test, and in cases of doubt this measure should be adopted.

Lymphatic leukæmia may prove a difficulty, but here blood examination at once reveals the condition. This disease is to be suspected if most of the glands of the neck are enlarged, if they are of an even size, and if no focus of infection can be discovered. A further examination reveals lymphatic enlargement generally, the spleen also participating. A case occurred at the Children's Hospital last year. I did not see it till on the operating table for removal of the tonsils for enlarged glands. On examining these glands they were generally enlarged in the neck, and in the other regions of the body. The spleen was readily palpable, and the case was sent to the medical ward for blood examination, which when carried out showed the changes characteristic of leukæmia. No operation was performed on this case, which emphasises the importance of a thorough examination before operation. The disease proved fatal in a few weeks. It is probable that some of the sudden deaths under anæsthesia in children are due to such a condition, which has been overlooked.

More rarely certain cysts of the nature of *hygromata* may cause some difficulty. These are single and no infective focus is present, while the tumour is found most frequently in the posterior triangle.

The general treatment of tuberculous glands is the same as for tuberculosis elsewhere, and those cases do best which are treated according to modern methods. In all cases the local source of infection must be found and removed. Following this, if the glands are in the early stage, they will in all probability subside and local treatment is unnecessary. In doubtful cases, where the gland or glands are as large as a walnut and still not softened, I wait for three months to see the effect

of general treatment. The glands are carefully examined during this period. At the end of this time if the glands are larger removal is advised, while if they are smaller or stationary they are watched for a further period of three months before surgical treatment is contemplated.

When one large gland and many smaller ones are present, the small ones frequently disappear after such a period, while no change takes place in the large gland, which from its size one knows will contain a large caseous area. I recommend a removal of this large gland, as it is liable to cause further infection and is not likely to subside.

In some cases where abscess formation is present, if the skin is already implicated, I incise the abscess and scrape out its contents. It is allowed a short time to heal up, and then this scar, together with the deep gland, is removed. If the abscess has not involved the skin, then it may be dissected out from the subcutaneous tissue, and by opening the deep fascia the caseous gland can be removed. Whatever the stage of the gland, even if an abscess requires immediate incision, the primary focus is removed before the glands are dissected out, where this is necessary.

I have not had a very extensive experience of tuberculin in these cases, but I have yet to be convinced that it exercises much influence on tuberculous glands. When the glands are extensively caseous I believe there is only one treatment, and that is surgical removal.

Before removal of tuberculous glands is advised, a general examination should be made to exclude other foci in the body. Although tuberculous cervical glands are a localised infection from the tonsil, the tubercle bacilli present in the milk may equally well have infected the respiratory or digestive tract, and there may be extensive lesions in these parts in addition to those at the entrance to these tracts.

Briefly detailed, the routine is as follows when a child presents itself with tuberculous glands. If the tonsils are the infective focus, the child is first referred to the dentist to have any carious teeth removed. When the mouth is clean the tonsils are enucleated, and when the throat is healed general treatment or operation is carried out according as it is necessary.

Summary.

(1) The site of an infective focus can be determined from the particular group of glands enlarged.

(2) The pharynx and naso-pharynx are the commonest sites from which the tubercle bacillus gains entrance.

(3) The tonsils are frequently tuberculous.

(4) A tuberculous tonsil may be enlarged, more commonly it is small and hidden, and very liable to be overlooked.

(5) Enucleation of the tonsil is essential if this is the infective focus.

(6) The supply of tubercle bacilli comes from tuberculous cow's milk.

(7) If the infective focus is removed the tuberculous glands may undergo a natural cure.

(8) If caseation has progressed far in a gland, complete resolution is almost impossible, and if the disease fails to improve on treatment, removal of the gland should be carried out.

(9) A rapid enlargement or softening of a gland means that a subcutaneous abscess has formed, and that scraping will not cure it.

(10) The gland causing the abscess lies under the deep fascia and may be quite small.

(11) Prevention is better than cure. Operations are difficult and should only be undertaken by a skilled surgeon, while recurrences, even after extensive dissections, are not unknown.

(12) Tuberculous neck-glands are a local disease, but are often associated with tuberculosis elsewhere in the body.

(13) The results of treatment may therefore be disappointing, and this emphasises the necessity for general sanatorium treatment.

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September 15th, 1913.

THE SIGNIFICANCE, TREATMENT, AND PROGNOSIS OF HIGH BLOOD-PRESSURE.*

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BEFORE considering the significance of high blood-pressure I will touch briefly on the controversy as to the reliability of the method in common use—the mercurial manometer with broad armlet. Is the reading afforded by the manometer a true measure of the arterial pressure, either systolic or diastolic? How far do clinical observations lend support, on the one hand, to the views of Russell (11) that the instrumental reading represents the true lateral pressure within the vessels plus the resistance offered by the vessel-wall, the latter factor being so increased by arterial hypertonus as to account largely for the high readings obtained; or, on the other hand, to the views of Hill (8) and his co-workers, that the readings afford a true measure of the systolic blood-pressure, the resistance offered by the vessel-wall being too inconsiderable to affect the result appreciably. Clinically, in this regard, four types of cases are met with:

(1) *The atheromatous type*, met with chiefly in old people, in whom the arteries are involuting, visibly and palpably tortuous, with lengths or patches here and there of calcareous thickening, separated, it may be, by lengths of soft-walled vessel. In these the pressure may be normal or low, but is not infrequently high, though nothing like to the same extent as in the diffuse form of arterio-sclerosis. Occasionally different readings may be obtained from the right and left brachials respectively, the one being more calcareous than the other, and failing to be completely compressed by the armlet. In a series of senile cases in a workhouse infirmary an examination of the blood-pressure in twenty-five men, of ages ranging from 65 to 89, the average age being 75, all of whom had arteries sclerotic in various degrees, showed a difference in the systolic pressure on the two sides in eleven cases—the maximum difference being 25 mm. Hg. Similarly, in a series of sixteen women, of ages ranging from 61 to 89, averaging 74 years, there was a difference in systolic pressure on the two sides in five cases, the maximum difference being 30 mm. Hg. This source of error may be to some extent guarded against by taking readings from both brachials, and if necessary from both forearms, from which a fairly accurate estimate may be formed of the true blood-pressure. If the whole of the brachials and radials are hard and pipe-like I doubt if a true measure of the blood-pressure is ever derived from the armlet manometer.

(2) *The diffuse arterio-sclerotic type*, with thickened vessels, firm but not necessarily hard, uniform and not patchy. The manometer almost always gives a high reading, probably in most cases approximately correct. Corroborative evidence of long-continued high pressure is found in the heart and elsewhere. In those cases, occasionally met with, having a systolic blood-pressure apparently enormously high, incomplete obliteration of the vessel taking place with an armlet pressure of 300 mm., I am inclined to doubt the correctness of the method, and to attribute the high reading to imperfect closure of the vessel owing to thickened walls. Co-existence of the nodular and diffuse forms of arterio-sclerosis would furnish an explanation. It seems unlikely that such a pressure, if truly registered, could be tolerated for long without disaster.

(3) *The hyperpietic type*, in which the blood-pressure is moderately high, the vessels full and tense, but not firm or hard, and the heart as yet undamaged. In these the rise recorded by the instrument may be exaggerated owing to hypertonus of the vessel-wall, relaxation of which leads to a speedy fall. No evidence of high pressure is obtainable from the heart and vessels, the mischief not having lasted sufficiently long to have caused gross changes; but evidence that the pressure is really above normal is to be found in subjective symptoms. If the hypertonus endures, sclerotic changes set in and the high pressure becomes permanent.

(4) *The "neurotic" or "functional" type*, generally seen in women, but by no means uncommonly in men. The vessels are soft, apparently normal to the touch, and to the finger is conveyed the impression of a normal or low blood-pressure. On taking the manometer reading the systolic pressure

* Read at the Royal Society of Medicine.

is found to be above normal, sometimes to a considerable extent. The diastolic pressure, however, is not raised in the same proportion. The heart shows no displacement of the apex-beat, nor evidence of hypertrophy by percussion, but the sounds are unduly forcible and the pulse-rate almost invariably raised. Undue apprehension at the moment of examination accounts in some cases for the disturbance; and when familiarity is engendered by repeated subsequent examinations the blood-pressure may be found normal. The rise in systolic pressure is merely temporary and is due to increased cardiac activity. But there are other cases of the nervous type, having no signs of arterio-sclerosis nor cardiac hypertrophy, in whom the blood-pressure, both systolic and diastolic, is constantly high, apart from transitory nervous excitation. Does the manometer give in these cases a true reading or an exaggerated one, due to spasm of the vessel-wall? I believe the reading to be true, and the explanation of the high blood-pressure to be that the pressure is set, as it were, at a higher level, and is the expression of a more rapid rate of tissue life per unit of time. The condition is capable of differentiation from hyperpiesis, and may not unjustly be termed "functional" high pressure as opposed to organic.

On the whole, then, with the exceptions stated, the method must be regarded as reliable and as furnishing a useful clinical guide. It is difficult to reject entirely the conception of arterial hypertonus giving rise to exaggerated readings, but inasmuch as the pressure is probably always above normal in these cases, though not perhaps to the extent shown by the instrument, and inasmuch as continued hypertonus may ultimately lead to sclerosis, the need for treatment arises, and therefore the error is not of much account.

It is a matter for regret that no entirely satisfactory means has yet been devised for measuring the diastolic pressure. Determination of the point of maximum excursion of the indicator is subject to a wide personal equation. A graphic record by Erlanger's or by Gibson's instrument is impracticable in ordinary clinical work. Further, there is the uncertainty as to the exact interpretation to be placed on the pressure taken at the point of maximum oscillation. The importance of the measurement is manifest, for though the systolic pressure may be high, the stress on the heart and vessels is much less if the diastolic pressure be relatively low.

In the cases referred to subsequently the figures given for the diastolic pressure are approximate, and represent the pressure taken at the point of maximum oscillation of the indicator.

The Significance of High Blood-pressure.

I am disposed to think that during the last few years, as one result of the attention drawn to the subject, the symptom of high blood-pressure has been regarded in rather too serious a light, not only by the profession generally, but also in the mind of the laity, to whom imperfect, hazy knowledge has filtered, leading to the usual untoward consequences. Many a patient has been unduly alarmed, many have been unnecessarily treated, and many are by no means the better for our treatment of this condition. I do not wish to seem pessimistic, but to sound a note of caution as to excessive and unnecessary treatment. During the last five years my attitude has become one of greater optimism towards the subjects of high blood-pressure, and of greater pessimism as to the wisdom or utility of energetic treatment in a large proportion of them. Given a patient with a high blood-pressure, the following questions arise:

(1) Is the pressure above the physiological limits? From the observation of a large number of workers it may be taken, in general terms, that up to the age of forty an enduring pressure (systolic) of 150 mm. Hg., and up to the age of sixty or over an enduring pressure of 160, may be regarded as pathological. Differences in size, weight and physique must, of course, be taken into account, a man of 20 st. usually having, other things being equal, a higher normal pressure than one of 11 or 12 st. Also it must be borne in mind that even high pressures may show considerable diurnal variation.

(2) Is the high pressure temporary or permanent? In a case showing no obvious signs of sclerosis repeated observation may be necessary, and the results of treatment gauged, before it is possible to determine whether the high pressure is due to transitory causes or is the expression of a permanent condition.

(3) If permanent, is it "functional," or organic? The determination of this point rarely gives rise to difficulty; most cases are easily relegated into one

or other group. Occasionally the problem may be hard to solve. Tactile examination of the vessels, the condition of the heart and kidneys, together with evidence of the neurotic temperament derived from general observation, and inquiry into the personal history, will usually suffice for a distinction to be made.

(4) If "functional," does it require treatment at all? In the large majority of instances I should say no. Here are brief notes of a case of functional high pressure that I have watched for some years. The high pressure has not given rise to any symptoms, nor does the patient seem the worse for it in any way.

CASE 1.—Mrs. P—, æt. 70 years; first seen in 1902 (then æt. 59 years); highly strung, nervous woman; suffers from mild arthritis; vessels, heart and kidneys sound. Blood-pressure record as follows:

Year.	Blood-pressure.		Pulse-rate.
	Diastolic.	Systolic.	
1902	—	180	96
1904	—	160	90
1905	—	150	96
1906	120	150	96
1907	140	170	94
1909	130	160	88
1910	130	160	96
1911	110	155	84
1912	120	170	84

No symptoms developed in consequence of the high blood-pressure. The systolic pressure is not unduly high, but it will be noted that the diastolic pressure is relatively low, to which fact, no doubt, she owes her immunity from trouble. Formerly when cases of this type came under observation I made an attempt to reduce the blood-pressure by treatment—diet, baths, drugs, etc.—with the result that when the pressure did fall they were by no means so well as when it was at their normal high level. Interference in these cases was unnecessary, though happily not prolonged sufficiently to become harmful. When left alone they keep in good health—apart from the minor ailments which led them to consult me. They are typical of many, from whom I have learnt that masterly inactivity is sometimes wiser than fussy interference.

Here is an example in a highly strung, nervous man who was sent to me last year as a case of arterio-sclerosis with high blood-pressure requiring energetic treatment.

CASE 2.—Mr. O—, æt. 50 years; good health all his life; no illnesses; has led an umblemished life; always been highly nervous and excitable; suffers from insomnia. His blood-pressure had been taken some months previously and had been found high, and the fact had been incautiously communicated to him. He had been treated by strict diet and a vigorous course of iodides and nitrites. His complaint was that he felt much worse while taking the medicines than without them. On examination his blood-pressure was 120/180, pulse-rate 104; heart not enlarged; a faint systolic bruit at apex and aortic areas; sounds unusually loud; vessels soft and uniform. Urine: specific gravity 1020, no albumen. The diastolic pressure, it will be noted, was low relatively to the systolic. There was no evidence of arterio-sclerosis, and the case was a typical one of "neurotic" high blood-pressure. He was reassured, his medicines were stopped, and his diet restored, which, with a course of bromide and valerian, speedily enabled him to regain his normal state of health.

Do these cases of what I have ventured to call "neurotic" or "functional" high blood-pressure live to the average term of life, or is it their tendency to be short-lived? Inasmuch as they live at a more rapid rate physiologically than the phlegmatic, low-pressured individual, they tend to wear out the quicker, but I am by no means convinced that their long-continued high pressure is of necessity a prominent factor in their premature decay. The interesting point is that they do not, as a rule, show signs of cardio-vascular degeneration, nor are they prone to die from vascular catastrophes. This may be explained by the fact that their diastolic pressure is low relatively to the systolic, and their vessels, therefore, are not in reality called upon to bear an excessive continuous stress; the stress is merely intermittent. Herein is emphasised the importance of not relying on the systolic pressure alone but of estimating the diastolic as well, and of considering the relation between the two. In a case, then, of high systolic pressure, with relatively low diastolic, and with the signs of cardio-vascular degeneration absent, no treatment (*ad hoc*) is as a rule required, and the prognosis is good.

(5) If organic, how far is treatment desirable,

and if desirable, how far is it of any avail? In the first place, let us consider what we are called upon to treat. We make certain assumptions which are more or less capable of proof.

(*a*) That the cause of the high tension is the presence in the circulating blood of toxins of chemical or bacterial origin.

Proof of this is afforded by the action of adrenalin and of pituitary extract; the effects of the poison of specific infection, such as syphilis, and the infective fevers, notably typhoid; the work of Metchnikoff on the bacterial toxins formed in the alimentary canal; the more recent researches of Bain (2) and others, showing that pressor bases found in the urine and derived from the putrefaction of proteid in the bowel are instrumental in causing a rise of blood-pressure. Since Josué demonstrated that arterio-sclerosis could be produced in animals by the hypodermic injection of adrenalin, the suggestion has been made by Sajous (12) that toxæmia of various kinds may give rise to arterio-sclerosis, not by the direct action of the particular toxin concerned, but indirectly through stimulation of the adrenal glands to excessive secretion. Cannon, Aub and Binger (4) showed that nicotine in small doses caused, in cats, an augmentation of adrenal secretion. Gilbert, Petit, Wybaux and others have shown that the infective fevers produce adrenal over-activity, sufficient to cause marked hypertrophy of the glands. Twenty years ago Abelous and Langlois showed that one of the functions of the suprarenal glands was to destroy the waste products of muscular work, and the suggestion is not unreasonable that prolonged over-activity of the glands thus induced may be the essential cause of the resultant arterio-sclerosis. From his experimental work G. von Anrep (1) concludes that every rise of blood-pressure brought about by the agency of the nervous system involves the co-operation of the chemical mechanism represented by the adrenal bodies.

(*b*) That the long-continued high tension leads to hypertrophy of the vessels and ultimately to sclerosis.

Proof of this is afforded by the morbid anatomy of vessels examined after various degrees and durations of high pressure. Or the problem may be viewed in another light, according to the suggestive theory advanced by French (7), who considers the toxin theory of arterio-sclerosis insufficient. He points out that sclerosis of the visceral vessels is constant in cases of prolonged high tension, and supposes the sequence of events to be: sclerosis of the visceral vessels resulting from excess of food plus sedentary habits; diminished function of the viscera; high tension of the peripheral circulation established compensatorily to ensure an adequate blood-supply to the internal organs; peripheral arterio-sclerosis following on long-continued hypertension.

(*c*) That we can control or limit the supply of toxins.

In so far as it applies to toxins generated in and absorbed from the alimentary canal, this assumption is in part true. By modifying the diet, diminishing fermentation, and promoting excretion, we can limit the supply of toxins. In so far as it applies to other forms of toxæmia, for example the infective diseases, we are powerless, in the present state of our knowledge, to prevent the consequences of infection; prevention of infection alone is possible. Similarly with regard to mental or physical stress; if this acts, as has been suggested, through stimulation of an excess of adrenal secretion, we have no means of limiting the amount produced except by lessening the work, at a time, probably, when irreparable mischief has already been done.

(*d*) That we can influence vessels already affected by the morbid process.

The proof of this is wanting except with respect to iodides in syphilitic arteritis. That iodides will cause the absorption of fibrous tissue in other forms of arterio-sclerosis the proof is unconvincing. The same applies to decalcifying agents, such as citric acid and the citrates; also to fibrolysin, which has been occasionally used in this condition. Even were it possible to effect the partial absorption of hypertrophied tissue in the vessel-walls, it is, to say the least, doubtful how far such a result would be salutary when we consider that the vessels have to support an increased pressure that has become necessary for the efficient performance of organic function.

In the second place, we must consider what becomes of cases of high blood-pressure when left untreated. The longer I follow up these cases the more am I struck by the length of time they survive and continue in apparently good health. One example may be quoted of many:

Case 3.—Mr. B—, æt. 63 years; first seen in 1902 (then aged fifty-one); an active business man, leading a strenuous life; has had good health all his life; moderate in his habits, except that he has smoked to excess. His blood-pressure record is as follows: During the whole period he had no special treatment beyond an occasional very brief spa course, and throughout he maintained his unusually strenuous habits.

The patient continues to follow his business, though perhaps with somewhat diminished vigour. His pressure throughout has not been superlatively high and the morbid changes are but slowly advancing. He probably has several years of useful life yet before him.

Year.	Blood-pressure. Diastolic.	Systolic.	Pulse-rate.	
1902	125	170	84	Heart sound, not enlarged; vessels normal.
1903	120	160	80	" " " "
1904	100	135	80	" " " "
1905	100	130	80	" " " "
1906	125	170	84	Apex displaced outwards; systolic bruit at aortic area; vessels readily felt; urine 1015; no albumen.
1907	130	180	82	Heart further enlarged.
1908	120	160	78	Physical signs unchanged.
1909	135	180	90	Breathless; slight œdema of legs.
1910	130	175	84	Systolic bruit more marked; vessels firm; œdema increased; urine 1006, trace of sugar, no albumen.
1911	130	180	75	Developed chronic glaucoma.
1912	120	165	80	Physical signs unchanged except that the vessels are becoming harder.
1913	140	170	78	Systolic bruit, apex and aortic; urine, 2 per cent. sugar.

Again, a visit to the wards of any workhouse infirmary will convince one of the fact that these cases may live for many years without treatment, in spite of an arterial condition that may be described in some instances as truly appalling. In the series of cases already quoted, of the twenty-five men examined rather more than half showed typical involutionary or atheromatous sclerosis, the remainder belonging more to the type of generalised or fibrous arterio-sclerosis. It is difficult to separate them clearly as the two groups overlap, many showing both conditions, as far as could be detected by tactile examination. Their average age was 75; average systolic pressure 176, maximum 250, minimum 130; average diastolic pressure 120, maximum 160, minimum 95. The difference between the average systolic and diastolic pressures was 56 mm. Hg. A systolic pressure of over 200 was met with in seven cases, all of generalised sclerosis. A systolic aortic bruit occurred in seven cases, having an average pressure of 215. The urine in all cases was of good specific gravity, and contained no albumen. No case showed signs of hemiplegia either of hæmorrhagic or thrombotic origin. Of sixteen women, the average age was 74; average systolic pressure 179, maximum 260, minimum 135; average diastolic pressure 124, maximum 160, minimum 100. The difference between the average systolic and diastolic pressures was 55 mm. Hg. Three cases had a systolic pressure over 200; five cases showed a systolic aortic bruit, their average pressure being 193. Hemiplegia occurred in three cases. It may be objected that there is no evidence how long the high pressure had endured; but from the state of the vessels the process must have been one of many years. Also, that these represent the survival of the fittest in a vascular sense. This is true, and the probable reason for their survival lies in the interesting fact which emerges from the figures —that, although their average systolic pressure is high (176 in men, 179 in women), the diastolic is relatively low (120 in men, 124 in women). In other words, that the vascular stress has not been extraordinarily great. The conclusion, then, to be drawn from the foregoing is that the presence of a high systolic pressure need not necessarily fill us with undue alarm as it is not incompatible with many years of useful life.

In the third place, how far can we by treatment prolong life or avert disaster in these cases? It is difficult to institute a comparison between treated and untreated cases, for we have no means of knowing how long the former would have lived if untreated. In the following example of declared arterio-sclerosis it is not irrational, however, to assume that life has been prolonged by suitable treatment.

CASE 4.—Miss P—, æt. 58 years, seen ten years ago, when she complained of headache, numbness and tingling of the right hand, with some loss of power. She was found to have generalised arterio-sclerosis, the cause remaining undiscovered. The blood-pressure was 150/200; heart enlarged; no bruit. Urine: 1015, no albumen. She was put on a careful *régime* as to diet, exercise and work, which she carried out faithfully. Her drug treatment consisted of a long course of Trunecek's serum, periodic courses of iodine and the iodides, a bi-weekly mercurial pill, and occasional doses of nitrite of sodium, which always gave relief to her symptoms. During the years that followed her blood-pressure varied from 180 to 230 systolic, and from 150 to 180 diastolic, being never below the smaller figures. When it approached the higher level her old symptoms of headache, paresis, and paræsthesia would return, invariably on the left side and accompanied sometimes by anginal pain. She went about as usual and performed her duties in a modified degree, always leading a quiet life. The heart gradually enlarged further, and two years ago a systolic aortic bruit developed and the vessels became thicker and firmer. The urine gave an average specific gravity of 1012, with, latterly, a trace of albumen. The pulse-rate remained uniformly about 66. In August, 1912, she quite suddenly developed auricular fibrillation; the pulse went up to 120 and showed the characteristic irregularity; dropsy supervened and the urine became loaded with albumen. A long period of rest in bed, with digitalis, wrought a marked improvement. The dropsy disappeared and the pulse came down to 70–80, though still irregular, the auricular fibrillation continuing. Since then she has taken her optimum dose of digitalis every day and keeps fairly well, though unable to do much. The heart is struggling against defeat, and how long it will take to succumb it is impossible to say, but there can be little doubt that careful following out of the treatment prescribed has resulted in prolongation of life.

While the treatment of well-established cases of arterio-sclerosis is, in a sense, unsatisfactory, in that a cure is not to be looked for, that of hyperpiesis, if caught early enough, is very gratifying.

CASE 5.—In a former paper I related the case of a university professor, æt. 40 years, who was a good example of hyperpiesis, due to overloading of the viscera and a sedentary life of hard mental work. I saw him first in 1902, when his pressure was 145/190, reduced by treatment to 130/170. He altered his mode of life and followed out the *régime* laid down for him. His pressure subsequently has read: 1903, 120/150; 1904, 115/140; 1905, 100/135; 1906, 100/130. I saw him again in 1910 after he had had a nervous breakdown, from various causes, when his pressure read 90/105, and after a course of tonic treatment 100/120. In 1911 the pressure was 100/120. In 1912 he had a mild attack of articular gout, and his pressure was 105/130. Through all these years there has been no return of the high blood-pressure. I could parallel this case with many others, but as they all follow closely the same lines their narration would be tedious.

An attempt may now be made to answer the question, how far is treatment desirable? and if desirable, how far is it of any avail? In the early cases of hyperpiesis treatment is imperative and usually attended by excellent results. In declared arterio-sclerosis general measures should always be recommended, designed to limit the production of toxins and to facilitate their excretion, with the view of delaying the inevitable progress of the disease; together with suitable modifications as to mental work, stress, exercise, and so forth. If drug treatment—with iodides, decalcifying agents, nitrites, and the like—be attempted it should be with caution; bearing in mind that a middle course must be steered between the Scylla of disaster from cerebral hæmorrhage on the one hand, and the Charybdis of cardiac defeat on the other. That unwise attempts to avert the former may, by over-drugging, precipitate the latter is no unreal danger. A fairly trustworthy guide may be found in the statements of the patient. If he feels the better for the reduction of pressure consequent upon drugs they may be continued; if he complains, as so many of them do, that he feels worse

when taking the medicines, it is well to discontinue them, bearing in mind that the gradually acquired high pressure has become essential for the efficient performance of the circulatory functions under the altered conditions.

What should the treatment be? From the foregoing it will be gathered that, in my judgment, far greater reliance should be placed on general measures than on drug treatment. Diet stands first; the intake of nitrogenous food should be lessened and the total bulk of food reduced. Alcohol should be limited or forbidden and abundance of plain water taken apart from meals. Next in importance stands elimination; the output of waste products must be promoted by all excretory channels, bowels, kidneys, and skin. Intestinal putrefaction, and the consequent absorption of toxins, must be prevented or lessened. Exercise, graduated to the needs of each case, must be enjoined and free action of the skin promoted. Mental work and stress should, as far as possible, be regulated. That mental stress and overwork are potent factors in inducing high blood-pressure there is abundant evidence to show. In my experience the mentally hard worked, harassed, free-living town-dweller is much more prone to suffer from generalised arterio-sclerosis than the physically hard-worked, plainer living out-door worker, who develops, rather, the focal atheromatous form. In this connection I may quote an instructive case under my observation.

Case 6.—Miss B—, æt. 30 years, came under my care in 1905 suffering from neuritis. Her blood-pressure was 80/100; in 1906, 85/110. In that year her circumstances changed, and since then she has, from various causes, led a life of continuous mental stress and worry, which has become more acute during the last two years. Her pressure record reads: 1907, 100/120; 1908, 110/135; 1909, 105/125; 1910, 105/125; 1911, 110/145; 1912, 120/150. The pressure is slowly mounting and her vessels are becoming unduly palpable. A permanent systolic pressure of 150 at the age of thirty-eight is suggestive of commencing arterio-sclerosis.

Into the details of these general measures I do not propose to go; they will be familiar to you all. Venesection must be mentioned as a useful proceeding in many cases. About drugs I have not much to say. The iodides, used so largely in arterio-sclerosis, owe their effect largely, I believe, to their action in reducing the viscosity of the blood. Insufficient importance has been attached to increased viscosity as a factor in the production of high pressure and consequent arterio-sclerosis. Cheinisse (5) draws attention to this, and points out that in arterio-sclerosis iodide reduces the co-efficient of viscosity from 5 to 10 per cent. after a fortnight's exhibition. Whether iodides can effect the absorption of newly formed fibrous tissue in the vessels is, to say the least, highly problematical. Too prolonged or excessive use of them tends to induce loss of tonicity and to accelerate cardiac defeat. Of the questionable utility of fibrolysin and of citric acid and the citrates as decalcifying agents I have already spoken. The nitrites and the whole group of vaso-dilators should be used sparingly to combat symptoms. The cases that react to them are the early ones in which the sclerotic process has not progressed far. Advanced cases will hardly react at all. A reduction of pressure effected by dilators is often of temporary service; but to use them freely, to secure a continuous reduction, is to invite impairment of cardiac tonicity and premature heart failure.

In the later stages of cardiac defeat digitalis is invaluable. The work of Price (10) and others has effectually laid the bogey of the risk of giving digitalis in cases of high blood-pressure. It may be given freely without raising the tension. In cardio-sclerosis the reaction to digitalis is said to be less than in the rheumatic heart, but in my experience the reaction has been good, more especially in those cases where auricular fibrillation has set in. The reaction probably depends on the amount of healthy muscle tissue left. No hesitation need be felt in giving digitalis, in the optimum dose, for months or even years continuously.

The Spa Treatment of High Pressure.

In early cases of hyperpiesis incomparably the best treatment is that to be obtained at a good eliminatory spa. Removal of toxic material from the bowel by aperient water or intestinal lavage; stimulation of the liver; copious diuresis and diaphoresis; acceleration of metabolism by massage and massage baths; reduction of pressure by warm saline and other baths; exercise, freedom from business worries—all the desiderata are fulfilled. A

course repeated twice a year at first, then yearly, due attention being paid in the interval to preventive measures, will suffice for the cure of most cases.

In declared arterio-sclerosis periodic spa treatment is of great service, the aim in view being the prevention, if possible, of further advance rather than the cure of mischief already done. The injudicious use of hot baths, as of drugs, in these cases may induce loss of tonicity and premature heart failure. Of radium emanation in the treatment of arterio-sclerosis I have no experience. Diathermic treatment may be helpful in relieving spasmodic cramp and intermittent claudication. The high-frequency current is, in my experience, disappointing as a depressor agent. In the so-called "functional" or neurotic high pressures subthermal immersion baths, from 90° to 98° F., are useful to allay cardio-vascular excitability and to promote a reduction of pressure.

Prognosis.

What are the points that guide us in forming a prognosis in any given case of high blood-pressure? In hyperpiesis, if reaction to treatment is fairly rapid and satisfactory, the heart and vessels are probably as yet undamaged, and the prognosis is good. Recovery is permanent if a rational mode of life be followed. In declared arterio-sclerosis the following points help us:

Age.—Other things being equal, the younger the patient with a high blood-pressure, not reacting to treatment, the worse the prognosis.

Height of pressure.—Within certain limits, say from 170 to 200 systolic, the height of the pressure does not help us much in forming an estimate. The contest lies between pressure on the one side and heart and vessels on the other. A high pressure with strong vessels may not be as harmful as a lower pressure with weaker vessels; and inasmuch as by the methods at our command we can form at best but a very imperfect conception of the real state of the vessels (and especially of that indefinable thing, their quality), we cannot get much help to prognosis in this direction. In the higher ranges of pressure—250 and upwards—the prognosis is, of course, bad; and the higher the diastolic in relation to the systolic pressure the worse the outlook.

The state of the heart and vessels.—Estimation of the quality of the vessels and the extent to which they are damaged is necessarily imperfect. Beyond the aorta and the iliac arteries, when accessible to palpation, we can form little idea of the extent of *visceral* sclerosis. Even when the peripheral vessels are apparently hopelessly damaged it is surprising how long a case may last. For example:

Case 7.—Miss L—, æt. 75 years, seen in 1907. Slight hemiplegic attack; recurrent unilateral headache with hemianopsia; sensory phenomena in hands.

Year.	Blood-pressure. Diastolic.	Systolic.	Pulse-rate.	
1907	155	210	96	Heart enlarged; no bruit; vessels very hard, thin and cord-like.
1908	165	220	—	Anginal pain; breathlessness.
1910	170	220	88	Irregular from extra-systoles.
1912	175	230	84	Irregular; anginal pain worse.

During the five years, in spite of her advanced age and high pressure, her downhill progress has been very slow.

Too gloomy a prognosis as to the probable duration of life need not be given even when the heart has begun to show definite signs of defeat. Cases 3 and 4 narrated above are examples in point.

Pulse-rate.—In a case of high blood-pressure the slower the pulse-rate, generally speaking, the better the prognosis. A persistent pulse-rate of 90 or over is of serious import.

The urine.—The presence of a trace of albumen in a urine of good specific gravity is not of much moment. Far more important is the specific gravity itself; if that be constantly low the prognosis is worse. A sudden increase in the amount of albumen is a bad sign.

Taken singly, these signs may not afford much help in prognosis; when more than one are present, and especially if all, the outlook is very bad, as the following case illustrates:

Case 8.—Mr. C—, æt. 44 years, schoolmaster. Previous history good; no illnesses; has led a very strenuous life mentally. Early in 1911 he had a slight hemiplegia with aphasia from which he made an almost complete recovery. His pressure then was 170/250; pulse 108; heart much enlarged; systolic bruit at apex and aortic; vessels very full

and firm. Urine, 1010, trace of albumen. He was carefully treated and every precaution taken, in spite of which his pressure rose further to 260 at the end of 1911. All the signs mentioned as of grave import were present, and in consequence a bad prognosis was given. Early in 1912 he had a severe cerebral hæmorrhage and died.

Lastly, in giving a prognosis it should be borne in mind that the subjects of arterio-sclerosis are ill fitted to stand the strain of any acute intercurrent disease.

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September 15th, 1913.

CLINICAL PATHOLOGY. By P. N. PANTON, M.A., M.B., B.C.Cantab., Clinical Pathologist to the London Hospital. 13 plates (11 coloured) and 45 illustrations. London: J. & A. Churchill. Pp. 446. 12*s.* 6*d.* net.

This work comprises laboratory investigations, whether chemical, histological, or bacteriological, bearing on the diagnosis and treatment of disease. As an adjunct to clinical medicine it is much more elaborate than most of the text-books on clinical pathology. The first section is devoted to the blood, methods of examination, parasitology, etc., and includes a most interesting chapter on blood changes which occur in children. The author thinks it is still a moot point whether the splenic anæmia of children (von Jaksch's anæmia) is a distinct clinical entity or a condition secondary to rickets or congenital syphilis. There can be no doubt that some cases of so-called pernicious anæmia in children belong to this class, an error which could instantly be eliminated by a blood-film. The second section is devoted to bacteriology and its methods, vaccines, anti-sera, preparation of culture media and staining reagents. The third section is devoted to puncture fluids, pleural, pericardial, peritoneal. In regard to the so-called malignant cells the author states that these, whether sarcoma or carcinoma, are frequently described, but practically never identified, as the sarcoma-cell is confused with the small lymphocyte, and the cancer-cell with the endothial. He then adds—"It has not been sufficiently insisted on that a carcinoma or sarcoma-cell floating free, has no distinguishing marks by which it can be identified." The fourth section is an exhaustive one on the subject of the urine, including special investigation and its bacteriology.

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A LECTURE

ON

CHRONIC APPENDICITIS.*

By JAMES SHERREN, F.R.C.S.,

Surgeon to the London Hospital.

THERE is still much uncertainty and misapprehension with regard to the diagnosis of chronic appendicitis and its relation to disease in other parts of the abdomen. For this reason I venture to address you on such a well-worn subject.

Pain localised to the right iliac fossa is popularly considered as *the* symptom of appendicitis. Such limited pain owns many causes, but is rarely due to the appendix.

I am using the term chronic in its clinical sense, including those cases which were at one time called "recurrent" and "relapsing," excluding those in which the illness is an acute one confining the patient to his bed. Many other definitions of chronic appendicitis have been given, for example, the pathological. Cheyne and Burghard,† in their text-book of surgical treatment, say: "Pathologically there is only one form of simple appendicitis which may be called chronic, namely *obliterative appendicitis*. Here there is a progressive destruction of the mucous membrane and ultimately complete obliteration of the lumen. This usually begins about the tip and gradually extends along the tube, but it may spread irregularly, forming strictures in the canal, shutting off spaces still lined with epithelium." This is a very common form that I am accustomed to term the atrophic or fibrotic. The symptoms associated with it are often vague, and are by no means always due to the appendicular condition. The large succu-

* Delivered at the London Hospital.

† Cheyne and Burghard, 'Manual of Surgical Treatment' (1913 edition), vol. iv, p. 404.

lent appendix in which there is a thickening of all the coats, often with stricture and retained mucus or pus, is the type responsible for the painful manifestations of the disease.

In 1902 Riedel,* I believe, first pointed out that a healthy appendix is practically never attacked by acute symptoms. In a recent lecture † I laid stress on this in the following words:

"Even that extremely common complaint, acute appendicitis, does not so often come absolutely unheralded by previous symptoms as we were at one time accustomed to think. In many cases careful inquiry will elicit the fact that there have been several attacks of abdominal pain passing off in a few hours, and thought little of by the patient before the acute and perhaps lethal one. In other cases attacks of flatulent dyspepsia, which may or may not be accompanied by pain in the right iliac fossa, should be a warning that all is not well with the appendix. In cases such as these a timely visit to the surgeon would not only save many lives, but would shorten the period of incapacity, and enable the operation to be carried out at a convenient season after necessary arrangement of business and social affairs.

"It is by no means an uncommon experience to operate on a patient with acute appendicitis who has been treated a considerable time as 'dyspepsia,' 'gastric or duodenal ulcer,' and diagnosed at the onset of acute symptoms as perforation of a peptic ulcer."

It is not, however, from the standpoint of the prevention of acute attacks, important as this is, that I am considering the subject this afternoon, but as a cause of abdominal discomfort and its association with disease in other parts of the abdomen.

I first wish to direct your attention to pain in appendicitis—in the initial stage of the acute attack; it is generalised and later shifts to the right iliac fossa. In chronic appendicitis I have been in the habit of teaching that the pain is not in the right iliac fossa, and if here, its principal seat is elsewhere. Less would be heard of failure to relieve symptoms by the removal of the appendix if we were sceptical of the sole appendicular origin of pain which is *limited* to the right iliac fossa.

These cases are more common in women and are usually associated with chronic constipation, often with visceroptosis. They often belong to the group of case in which at the present time the treatment cannot be considered finally settled in which a mobile cæcum, Lane's ileal kink, Jackson's membrane, are found. In other cases, again, particularly in men, ureteral calculus is present, and the cause of the attacks. Recently I have had three examples of this under my care. In one the appendix had been removed at another hospital without curing the attacks, which were due to a stone in the pelvic portion of the ureter. The other two I was asked to see and remove their appendices. The one suspicious feature in each case was the starting-point of the pain in the right iliac fossa and its limitation to this region. I was prevented falling into error by X-ray examination, which revealed calculi in the pelvic ureter in both cases.

More recently I have had two men under my care, both of whom had had pain limited to the right iliac fossa. Before operation X-ray examination revealed nothing abnormal and urine was normal. I removed slightly adherent appendices, but both have returned with their old pains.

In none of the patients had urinary symptoms been marked; they were less in many patients with disease of a pelvic appendix.

Cases of chronic appendicitis may be divided into two groups: (1) with attacks of pain, (2) the dyspeptic type.

(1) This is a most important group, in which the patient complains of *attacks of pain* at irregular intervals with periods of perfect health usually free from any dyspepsia. The pain is in the umbilical or epigastric region, sometimes in the left lower abdomen, and is usually, though by no means invariably, also felt in the right iliac fossa. These attacks are the result of subacute exacerbations, or of sudden blocking of the lumen of the appendix.

If we are able to examine the patient during the painful period deep tenderness is usually present either in the right iliac fossa or *per rectum*, but it may be entirely wanting between the attacks. In a number of the cases typical superficial tenderness is present having the characters that I described in 1903. Vomiting may be present or absent, and little stress should be laid on this.

There may be symptoms in other organs due

* Von Langenbeck's 'Archiv für klinische Chirurgie,' 1902, vol. lvxi, p. 1.

† Clinical Journal, September 6th, 1911, p. 347.

to their irritation or implication in disease; thus the attacks may be followed by, or associated with, frequency and pain on micturition, also with rectal and perinæal pain, the latter of which I have known treated as the "rectal crises" of tabes. I will give examples of each.

The following is typical of this group. Last December I saw a man, æt. 35 years, who for seven years had been having attacks of abdominal pain sometimes associated with vomiting. The pain was epigastric or umbilical, never in the right iliac fossa. It often woke him in the early morning, and would last a few hours to two days. During one attack at least there had been tenderness in the right iliac fossa. At operation I found the appendix markedly thickened and strictured, lying adherent on the outer side of the cæcum.

The pain may be wholly left-sided. Several cases of this have come under my care. I believe that in these cases the appendix will be found in the pelvis. This was so in four recent cases, in two of which there was also frequency of micturition.

In October, 1912, I saw a girl with the following history: For eleven months she had been having attacks of abdominal pain and vomiting. The pain was always above and to the left of the umbilicus and associated with frequency of micturition. She had had no pain or discomfort after food. I saw her three weeks after an attack. Abdominal examination revealed nothing wrong. I was suspicious of attacks of obstruction in the ureter of the left kidney by an abnormal renal vessel. The urine was normal. Radiographs showed the outline of a kidney of natural size; no shadow was noted here or along the course of the ureter. Rectal examination was negative. I then examined her under an anæsthetic and cystoscoped her without detecting anything.

As it was impossible to be certain of the diagnosis I allowed her to leave hospital, giving her instructions to return during an attack. She returned December 30th. She said that two days previously she had had a severe attack, and on this occasion the pain spread from the left side to the umbilical region. The diagnosis now was clear, for rigidity and tenderness were present in the right iliac fossa. On examination *per rectum* there was tenderness high up and to the right. At operation I found the appendix hanging over the brim of the pelvis. Close to the cæcum it appeared normal, but beyond was much dilated; its peritoneal coat was roughened. The terminal ileum was normal. All other organs were normal. On cutting into the appendix a fibrous stricture was found an inch from the cæcum. Beyond this it was dilated; its wall was thickened and it contained foul-smelling pus.

A further example is the following: In November, 1912, I saw a man, æt. 29 years, who gave a history of attacks of abdominal pain extending over three years. The onset was sudden; pain in the lower left abdomen, with vomiting. Three attacks at intervals of a month, perfectly well between, and after the last attack remained well till November, 1911. Then attacks of pain in the epigastric region on the left side, very severe, sometimes waking him between 1 and 2 a.m., and accompanied by retching. During the whole time he had had slight frequency of micturition.

Abdominal examination was very difficult. I found the patient quite unable to relax his muscles. *Per rectum* nothing abnormal felt. The attacks of pain in the upper abdomen were suggestive of gall-stones. The urine was normal. Test meal, free HCl ·08, total acidity 44. Report of examination after bismuth meal was—"The stomach is seen to empty itself rapidly. The condition seen is suggestive of ulcer."

At operation I removed two calculi from the gall-bladder and drained it. Through a separate incision I explored the appendix region. I found the appendix running over the brim of the pelvis, closely covered by adherent ileum, making a firm mass. With great difficulty I separated and removed a large tense appendix. On cutting into it I found much thickening of its walls and a complete stricture an inch from its tip; beyond this was a cavity containing foul-smelling pus and a concretion. I have no doubt that the three attacks at the onset of the illness were appendicitis, the later one due to cholelithiasis.

When the appendix lies in the pelvis the attacks may consist of rectal pain, tenderness, and frequency of micturition.

In January, 1912, I saw a patient, æt. 57 years, who had been having attacks of abdominal pain since the age of fifteen. Until two to three years before I saw him these had been umbilical and not associated with vomiting, and only occasionally laying him up for a day or two. From this time

on he often awoke at night with perinæal and rectal pain, desire to pass urine, and pain on micturition; this was marked in all subsequent attacks. Deep tenderness was present over the lower right rectus, and *per rectum* a tender swelling could just be felt high up and to the right. At operation I found a thickened appendix adherent to the side wall of the pelvis and to the ileum. The mucous membrane in its proximal half appeared normal, but its wall was much thickened; its distal two inches was dilated to form a smooth thick-walled cyst containing muco-pus.

In the following case the pain was entirely rectal. In April, 1912, I saw a man, æt. 48 years, who for two years had been having violent attacks of epigastric pain, usually waking him at night—typical biliary colic. Six months before I saw him he had a sudden severe pain in the rectum, lasting about two hours and accompanied by retention of urine. He has had six attacks since then, all at night. These attacks had been diagnosed at first as rectal crises. Abdominal examination revealed deep tenderness in gall-bladder region, and tenderness high up *per rectum*. At operation, in addition to gall-stones, I found a much thickened appendix running right across the floor of the pelvis, very adherent, reaching to the right side. He entirely lost his symptoms.

To sum up, chronic appendicitis of the first type is associated with attacks of abdominal pain, usually epigastric or umbilical, occasionally wholly left-sided. The pain may shift from its first seat to the right iliac fossa, but this is not invariable, although if examined during an attack tenderness is usually present. Attacks occasionally come on after exercise, but between the attacks the patient is generally well and does not usually suffer from indigestion. When the appendix is pelvic, vesical or rectal symptoms may also be present. In this type of case the appendix is usually grossly enlarged, thickened, often strictured, containing mucus or pus.

During the past three years I have operated upon sixty-seven cases of this type, forty-three of whom were men. Of these eighteen had had indigestion, but in only one was it a prominent feature for which the operation was performed. In the others it was an incident which had to be inquired for and was only marked in three. In all it was of the flatulent type.

We now pass on to the second type of case, in which the symptoms are not those of attacks of abdominal pain lasting a few hours, but of discomfort of longer duration, situated in the epigastric region, often bearing a definite relationship to the ingestion of food, in some cases simulating, but never completely imitating, the symptoms of gastric or duodenal ulcer. Although it is fourteen years ago since Ewald* directed attention to this group of case under the name of "appendix larvata," it is only during the last three or four years, owing in great measure to Sir Berkeley Moynihan, that the part played by the appendix has become recognised.

During the last three years I have operated upon sixty-three patients (twenty-five of whom were women) for "indigestion." By this I mean that the diagnosis of the cause of the indigestion could not be made with certainty before operation, and the upper abdomen was first explored before a diseased appendix was found and removed. In forty-nine of these the appendix was of the atrophic obliterative type, often being very adherent. It is interesting to note that this is the type of appendix found so frequently associated with chronic ulcer of the stomach and duodenum. Thus in eighty-three consecutive cases of chronic gastric and duodenal ulcer with no history of appendicular disease I found this type in sixty-two, in thirteen only it was of the thickened type, in eight normal.

The symptoms may mimic either gastric or duodenal ulcer, but the clear-cut history which obtains in both those conditions is absent. Pain is usually felt in the epigastrium, occurring sometimes in irregular attacks, at others being present nearly every day. It bears no regular definite relation to food being felt, for example, an hour after one meal, immediately after or even during another. It may be referred to the right iliac fossa, where tenderness may be present, the pain on pressure being appreciated in the epigastric region. Nausea is frequent but vomiting uncommon, although hæmatemesis may occur.

The condition is confined to no age-period (youngest 18, oldest 67), but most of my patients were between the age of thirty and forty. The sex-distribution differs little from that of other types of

* Von Langenbeck's 'Archiv f. klin. Chirurgie,' 1899-1900, vol. lx, p. 80.

appendicular disease. Several of the older ones were sent to me as carcinoma of the stomach.

The following is an example: In June, 1910, a woman, æt. 55 years, was sent to me with the following history: In 1898 she suffered from "gastric ulcer" for two months. Another attack in 1904. For six months before I saw her she had had pain at irregular intervals after food, vomiting about once a day. Always constipated. For six weeks pain had been present in right iliac fossa. I found rigidity and tenderness in right iliac fossa. Test-meal free HCl ·1. Total acidity 52. At operation I found stomach, duodenum and gall-bladder normal. I then exposed the appendix, which was kinked and adherent. On cutting into it it was thin and fibrotic and its tip was becoming obliterated. She left hospital sixteen days later and has remained quite well.

A few months later I operated on a male, æt. 46 years, who had been treated for chronic gastric ulcer off and on for fourteen years. He was in bed for two months with his first attack and had never been really well since. His pain had been five to ten minutes after food, relieved by belching or by vomiting, but the latter was very infrequent. No night pain. A month before I saw him he had been confined to bed with a severe attack of hæmatemesis. At operation I found stomach and duodenum normal, gall-bladder normal. The appendix was tightly bound down on the outer side of the cæcum by adhesions. It was thin, fibrotic, and becoming obliterated at its tip. He has remained quite well.

Cases of this type could be multiplied. Of twenty-two cases operated upon over two years, the majority of whom had had indigestion many years, one cannot be traced, fourteen are quite well, six are much better and only one is unrelieved.

With regard to diagnosis. In the first group of case little difficulty is experienced in the large majority of cases. Attacks of general abdominal pain, particularly if referred after a period to the right iliac fossa, are nearly always of this nature. When the pain is wholly right-sided the appendicitis should be the last and not the first condition thought of. In males particularly ureteral calculus should be carefully considered, X-ray examination and cystoscopy with the passage of ureteral bougies not neglected.

The diagnosis of chronic appendicitis must not be made in women with constant aching pain in the right iliac fossa until every other possibility has been excluded. Operation is the last resource after other forms of treatment have failed. Abdominal exercises, the administration of aperients, particularly paraffin, which in many of these cases acts extremely well, and the wearing of an abdominal belt will often cure. Removal of the appendix alone often fails to relieve. When cure results from operation many adhesions have often surrounded the appendix, and it is the freeing of cæcum and ileum which has resulted in the cure.

In the cases in the second group diagnosis is often impossible. We can suggest before exploration that the appendix is at fault, but in few is it certain enough to neglect upper abdominal exploration.

With regard to examination by bismuth and test-meals, in the former vigorous contractions of the pyloric end of the stomach suggestive of ulcer may be noted, in other cases irregular contraction producing functional hour-glass stomach. From the latter important help is often given, for while very occasionally high acidity may be revealed suggestive of chronic duodenal ulcer, as a rule it is low, and frequently there is an absence of free HCl which is extremely suggestive. Thus, among 39 consecutive cases in which test-meals were given acidity free and total was diminished in 21, free HCl was absent in 15 of these, normal in 7, a little raised in 11, but only in 2 was it high. One free HCl ·22, total acidity 84, the other ·30, 93. I could find no difference in the type of appendix associated with differences in the test-meals.

The diminution of acidity often with absence of free HCl is an almost invariable finding in hour-glass stomach and a frequent one in gall-stones. The former may be excluded by the bismuth meal; the latter cannot, for the flatulent dyspepsia in the prodromal stage of gall-stones may resemble very closely that associated with chronic appendicular disease.

There is one test about which I wish to say a few words, that is, Bastedo's.* This consists of distension of the colon with air. Too great reliance must not be placed on this. I have found it positive in a woman upon whom I operated for chronic

* 'Amer. Journ. Med. Sci.,' July, 1911, p. 11.

gastric ulcer and removed her appendix. It was free from adhesions, and to the naked eye and microscopical examination appeared quite normal. I found it negative in two patients, from whom I removed thickened and strictured appendices containing pus.

The indigestion associated with chronic constipation (intestinal stasis) is often mistaken for that due to chronic disease of the appendix. It is rarely, however, that discomfort after food of the type met with in this condition is the only symptom; pain is nearly always complained of in the right iliac fossa, where there is tenderness and often the terminal coil of ileum can be felt. In both, however, there is usually hypo-acidity, often an absence of free HCl, and in both the bismuth meal may show irregular and vigorous peristalsis.

With regard to operation, whenever the abdomen is opened it should be through an incision as small as is safe for efficient working. Where the diagnosis is clear I am in the habit of removing the appendix through a small muscle-splitting incision. This can readily be enlarged if necessary by continuing the inward splitting of internal oblique and transversalis muscles, opening the sheath of the rectus and retracting it inward. If I diagnose the appendix as pelvic, or am uncertain of the diagnosis, or consider pelvic complication likely in women, I use Battle's incision.

In cases of appendix dyspepsia I explore the upper abdomen through an incision made over the right rectus muscle, incising its sheath and pulling it outwards. I never attempt to remove the appendix through this opening. Gentleness of manipulation, the avoidance of "pulling" are essentials to success in abdominal surgery; unless the incision is unduly lengthened these rules must be broken if an attempt is made to remove it through the upper incision. Again, unless adhesions are dealt with by sight damage is easily done. It is for this reason that I remove the appendix in these cases through a separate small muscle-splitting incision. In every case the incision must be made large enough to effectually examine the whole cæcum and terminal ileum.

September 22nd, 1913.

INTESTINAL ANGULATIONS AND KINKS ASSOCIATED WITH STASIS.*

By W. I. DE C. WHEELER, F.R.C.S.I.,
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THE profusion of recent literature on the subject of intestinal stasis produced by non-inflammatory intestinal adhesions and kinks, with all the varying and contradictory expert opinions put forward, renders it difficult to compile a communication in any sense complete. I will confine my remarks to personal experiences with twenty recent abdominal cases which illustrated conditions associated with the term intestinal stasis. Some or all of the pathological conditions which I mention later were present.

It is not so long since we were taught that intestinal kinks and adhesions other than inflammatory were a product of the imagination—"A dagger of the mind, a false creation." Those who were first to discount the possibility of the presence of kinks and adhesions have now taken refuge in the statement that every individual has adhesions and kinks, and they do no harm. These two contentions are obviously impossible to adjust or harmonise.

It may be now taken as an axiom that in a large percentage of abdominal cases, cases of gastric or duodenal ulcer, appendicitis, gall-stones, and allied conditions, including cystic ovary, there is present a varying degree of intestinal stasis associated with the presence of certain pathological angulations and kinks to be mentioned later.

In a certain proportion of the cases these conditions, *per se*, give rise to no symptoms whatever, in others the symptoms are slight or masked; in the remainder they are so marked that a novice could not be deceived as to their cause.

The connection between, say, duodenal ulcer and intestinal kinks and other pelvic lesions is in dispute; that there is a close connection is beyond doubt, and it has led Moynihan to remove the

* Read at the Royal Academy of Medicine in Ireland. Illustrations by courtesy of the 'Medical Press and Circular.'

appendix in most cases of duodenal ulcer, and Lane to abandon the operation of gastro-enterostomy except for the relief of pyloric obstruction. From the admitted association of various intra-abdominal lesions there is one practical lesson to be learnt—the fallacy of performing very rapid operations for the removal of a non-suppurating appendix. If the appendix is in an anatomical position, free from pathological adhesions, and lends itself to rapid removal, either the operation is not justified, or, more likely, the symptoms which called for the operation were due equally to other associated abnormal conditions. Surgery has been discredited by the persistence of symptoms after removal of an appendix by surgeons ignorant of the existence, say, of a Jackson's membrane or other abnormality. The presence of a surprisingly normal appendix in a patient who suffered from symptoms sufficiently severe to warrant operation should call for pause, and a careful search be made for abnormalities at the terminal part of the ileum, the ascending colon, and hepatic flexure, and if need be in all "four corners" of the abdominal cavity. The removal of an appendix in four or five minutes is easy of accomplishment, but does not savour of scientific surgery, and the diagnosis of "appendix dyspepsia" before operation in the light of recent advances in abdominal surgery can seldom be justified.

The following lesions were well marked in my series of cases, some in one, others in another; the presence of one abnormality did not necessarily mean the presence of another, although in several cases all the lesions to be mentioned were well marked and distinct in the same case. Thus, in some, appendicitis would be accompanied by a well-marked Jackson's membrane, a Lane's kink near the ileo-cæcal junction, and a mega-duodenum. In almost every case the terminal mesocolic kink (pelvic colon) was present, in some causing undoubted abdominal symptoms, in others doing no harm whatever.

Lane's terminal ileal kink was described as caused by a band first forming part of the under surface of the mesentery, and afterwards a distinct ligament which attached itself progressively to the ileum more and more distant from the mesenteric border. The band is shorter than the mesentery, thus causing a pucker or kink of the ileum, which

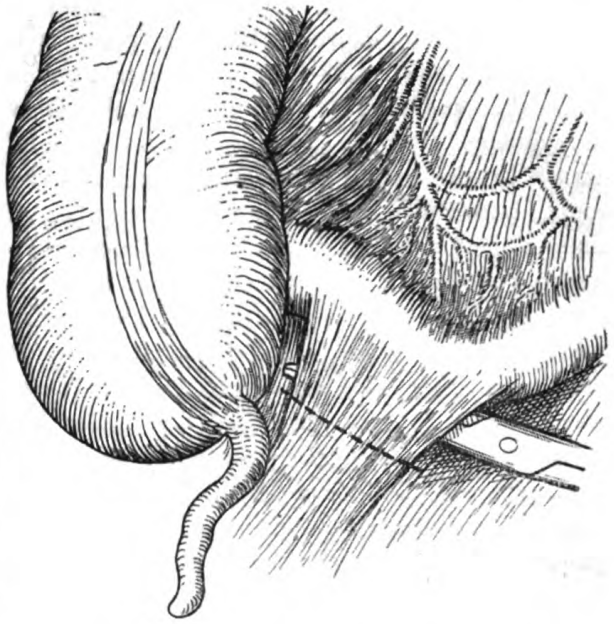

FIG. 1.—Diagram of Lane's terminal ileal kink.

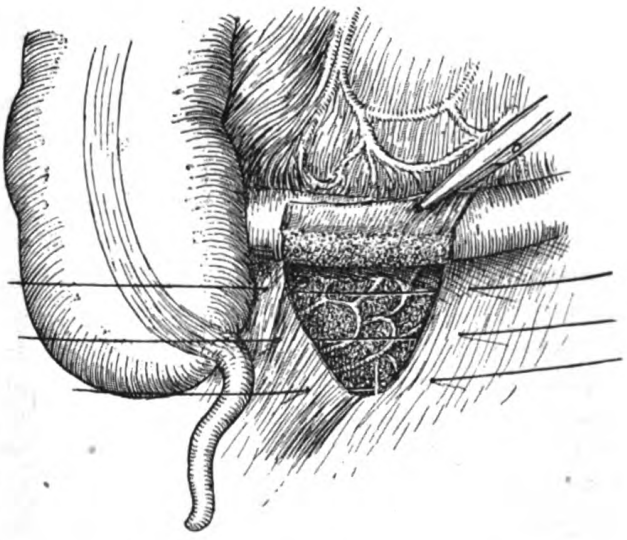

FIG. 2.—Lane's ileal kink relieved by plastic operation. The peritoneal flap held by forceps covers the raw surface of the ileum. The peritoneal edges are approximated by sutures. (Modified from Gray and Anderson.)

is, in addition, often rotated on its long axis owing to the attachment of the band to the anti-mesenteric border.

According to Lane, this and other intestinal kinks are due to "the crystallisation of lines of force" to prevent prolapse of the viscera in cases

of chronic constipation. Gray and Anderson do not accept the explanation, and point out that in both the lower ileal and lower sigmoid kinks the mesentery and mesosigmoid show no evidence of "the crystallisation of lines of strain" in the upper layers where most strain would be expected, and that in a number of cases of ileal kink there was no tendency whatever to a dropped cæcum, which, on the contrary, was well fixed. In my cases of ileal kink, which were comparatively few, I could not satisfy myself that the small intestine was much, if at all, dilated above the lesion as described by Lane, but in two there was distinct evidence of megaduodenum. This latter condition is produced not in any direct manner from the ileal kink, but by a similar adventitious formation at the duodenojejunal flexure, or to a band running across the duodenum just to the right of the superior mesenteric artery, as described by Murphy. Jackson s membrane has attracted considerable surgical attention during the past twelve months. It is a very obvious lesion found regularly in patients suffering from right-sided pain or discomfort with a loose, splashing cæcum, constipation, and some visceroptosis. It is present in other cases, but one soon gets familiar with a type of case where its presence can be foretold almost with certainty before operation. It is a thin, vascular veil, in which long, straight blood-vessels run a parallel course, compared by Jackson to an œdematous arachnoid. It is like a cobweb bag which is too small to hold the ascending colon without puckering it. It extends from the hepatic flexure to the cæcum, and may surround the colon on both sides of the anterior longitudinal band, merging below into the meso-appendix and pelvic peritoneum. At the hepatic flexure the membrane when lifted up on the finger often forms a formidable band binding and obstructing the colon (Fig. 3).

The better developed the Jackson's membrane, the more prolapsed and distended is the cæcum. This is an observation of importance, for it teaches that it does not assist in fixing a mobile cæcum, but, on the contrary, by sometimes producing a very acute kink at the hepatic flexure of the colon causes dilatation and ptosis of the cæcum and ascending colon. It is noteworthy in these cases that the transverse and descending colon are collapsed in marked contrast to the cæcum and

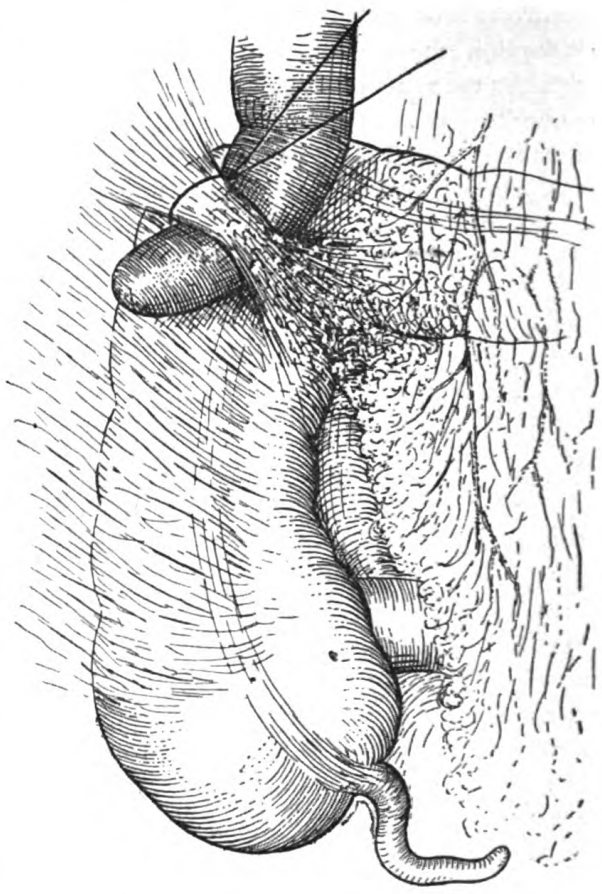

FIG. 3.—Jackson's membrane, showing the well-developed band merging into the omentum, obstructing the hepatic flexure and producing dilatation and ptosis of the cæcum. These cases are frequently referred to erroneously as "appendix dyspepsia." The bands are hooked up on the finger and divided from the flexure to the cæcum.

ascending colon. It is therefore more likely that Jackson's membrane is a cause, and not an effect, of intestinal stasis.

It is almost certain that Jackson's membrane is not inflammatory in origin. A study of the direction of the lymphatics proves it could not be a pericolic inflammation which had extended along the lymphatics of the subperitoneal coats. Furthermore, the blood-vessels run in a linear fashion, unlike those in inflammatory tissue, and no bleeding results from the general separation of the membrane, but, on the other hand, a sufficiently raw surface is sometimes left to necessitate the application of an omental graft to prevent subsequent adhesions. In young children Jackson's membrane is often well marked.

Mayo's theory of abnormal burrowing of the cæcum and lower end of the ileum during their descent from the liver under the parietal posterior peritoneum has been dismissed owing to the absence of such a process in the examination of a very large number of fœtuses.

Gray and Anderson's is the most fascinating explanation, and appeals to the imagination of those who have studied the structures at operations. They say the membrane is really the right margin of the great omentum. The omentum crosses the hepatic flexure and blends by physiological fusion with the posterior parietal peritoneum to the outer side of the colon. This fusion also takes place between the omentum and anterior wall of the colon near the longitudinal muscular band in the same way as the omentum finds attachment to the transverse colon in a corresponding position. During the descent of the cæcum the omentum crossing the flexure and becoming adherent to the parietal peritoneum externally gets pulled out, and is found in later life as the membrane already described. This theory is strengthened by the presence of fat, the linear arrangements of the vessels, and the site of attachment of the membrane to the colon. In several of my cases I could trace a direct and uninterrupted connection between the membrane and the omentum. This was specially noticeable above at the hepatic flexure, where Jackson's membrane often takes the form of powerful bands intermingled with fat, obstructing the onflow of the contents of the colon (Fig. 3).

In addition to the ileal kink, the hepatic kink produced by Jackson's membrane, the dilated duodenum already referred to, I have had one case of Payr's disease, where there was a well-marked kink of the splenic flexure, with dilatation and ptosis of the transverse and ascending colon, and collapse of the descending colon. It is, I believe, a comparatively common condition, but I have only seen it twice before, once in a case operated upon by a colleague before its significance was understood, and once with Mr. Gray, of Aberdeen. The kink is probably due to a shortened phrenocolic ligament.

In two thirds of my cases the lower sigmoid kink was present. So frequently is this condition seen that it is looked upon as almost normal by some authorities. All are agreed, however, that

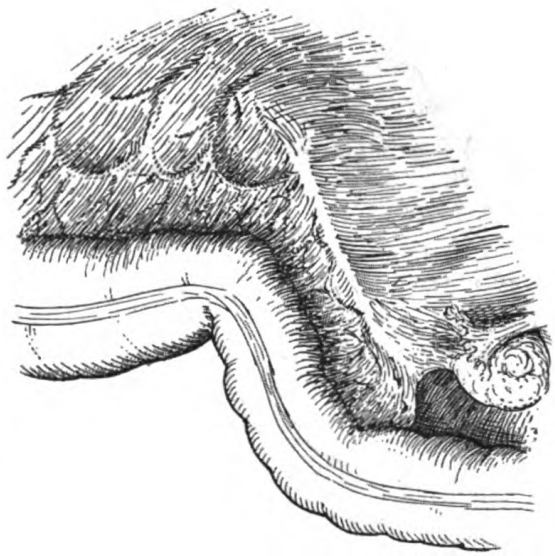

FIG. 4.—Kink of the pelvic colon, showing "Toldt's line" and implication of the left ovary. Obscure left-sided pain and constipation is frequently produced by this condition and relieved by operation. (Modified from Gray and Anderson.)

in certain cases it produces very marked constipation and left-sided pain with dilatation of the colon above. In many cases, on the other hand, it interferes with the bowel little, if at all. Here we can see a beautiful example of a kink produced by the excess of physiological fusion as described by Toldt (Fig. 4).

Gray attributes the formation of all kinks to excess of physiological fusion, and it has been shown that the formation of Jackson's membrane can be attributed to a like cause. While Gray believes enteroptosis is produced by the presence of obstructive kinks, yet the congenital theory can be made clear by another interpretation. When man assumes the erect position, in order to prevent a drop of all the viscera into the pelvis, Nature, by physiological fusion of the visceral and parietal peritoneum, produces the splenic and hepatic flexures, the mesentery and meso-colon, and all ligaments intended to suspend the viscera.

Assume we approach at birth towards our ancestral condition, and suffer from slight congenital ptosis, or assume that later in life ptosis is produced from familiar causes, then there may be peritoneal fusions in unusual places to ensure fixation as a provision of Nature against further prolapse.

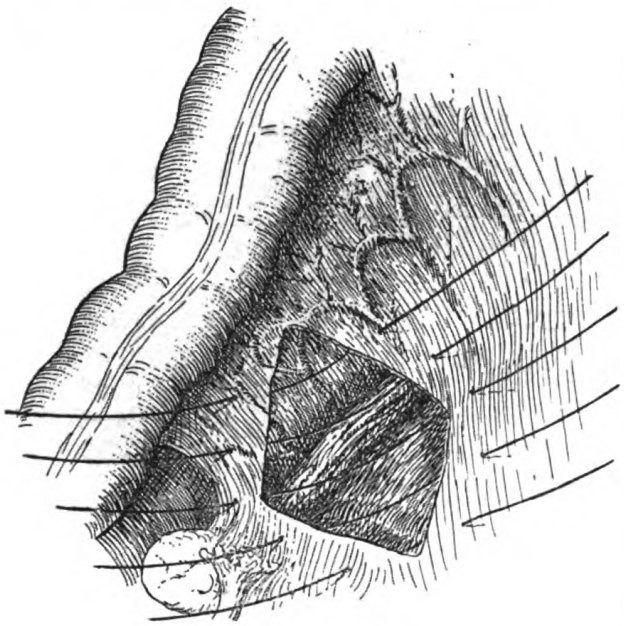

FIG. 5.—Kink of the pelvic colon relieved by plastic operation. (Modified from Gray and Anderson.)

To revert to the terminal kink of the pelvic colon, which is such a common condition, if we pull on the colon we can see the line of fusion between the outer side of the mesocolon and the peritoneum of the left iliac fossa. This line is known as Toldt's white line (Fig. 4).

In cases of chronic constipation, in the absence of other causes, the "undoing" of this kink, especially in enteroptosis cases, will have the effect of producing a regular daily motion, and a consequent great improvement in the condition of this miserable class of patient.

I have operated upon several cases for my colleagues, Drs. Lumsden and Holmes, with the result stated.

Left-sided abdominal pain with constipation may often be completely relieved by a plastic operation on this kink, and it should be remembered that the pain is often rendered more severe by implication of the left ovary and broad ligament in the line of fusion (Fig. 5).

The congenital theory is not upset by the contention that symptoms do not arise until adult life. We have only to think of such conditions as cervical rib and congenital hernia to see where the fallacy of such a contention lies. The nature of the operation required in each case is shown in the illustrations and requires no further description.

In two of my cases there were so many developmental adhesions and kinks that I short-circuited the ileum into the lower sigmoid—the operation recommended by Lane. (It is not possible, for anatomical reasons, to anastomose the ileum with the rectum as frequently stated.)

If the permanent results of the plastic work are as good as the immediate results the short-circuiting operation will become unnecessary, but in any event the operation, as often attributed to Lane, is not performed in the manner he recommends, and gives correspondingly bad results. The ileum must be completely divided and anastomosed low down below the sigmoid kink.

If the kink is not well marked it should be artificially produced to prevent reverse peristalsis—a complication which, in early days, rendered necessary a subsequent colectomy in a fairly large proportion of cases.

In my series of cases the symptoms were very varied. Left-sided pain and discomfort by a process of elimination was diagnosed in several cases as being due to a well-developed meso-sigmoid kink. These patients, usually females, were cured by operation.

Diffuse right-sided pain, tenderness, and discomfort in patients, with a mobile splashing cœcum and distended ascending colon, were found usually to present a well-marked Jackson's membrane and hepatic kink; the appendix often showed little or no signs of disease. The ileal kink was present in some of these cases, but absent in at least an equal number. One case diagnosed as duodenal ulcer proved to be a mega-duodenum accompanied by Jackson's membrane and ileal kink. The patient was a medical man, and presented all the signs and symptoms of duodenal ulcer. X-ray photographs supported this view, but on operation only the conditions mentioned were found. The patient was completely and immediately relieved by a plastic operation.

Two cases only were typical of auto-intoxication—there was staining and wrinkling of the skin, sweating in the axillæ and groins, enfeebled circulation with cold extremities, dulling of the mental faculties, etc. The complete short-circuit operation was performed. These patients are

tolerably well. One was done two years ago; the other has just left hospital, but still complains of abdominal distress.* The number of ailments directly caused by intestinal stasis are probably legion, but of the remote lesions, such as chronic rheumatoid arthritis, cystic breasts, and hypothyroidism I have no personal knowledge, and therefore I only mention them to dismiss the subject.

I am convinced, however, that many cases of rheumatoid arthritis in the early stages can be cured or relieved by the correction of intestinal lesions causing stasis, in a more certain manner than by the administration of intestinal antiseptics and other drugs.

X-ray photographs were taken of a number of the cases by Dr. Hayes to assist localisation of the lesion before operation.

I must acknowledge my great indebtedness to Mr. Gray and Sir Arbuthnot Lane for allowing me to see their operations and results in connection with this subject.

September 22nd, 1913.

Toxic Action of Salvarsan.—Brandenburg reports the case of a robust man of 38, who had had no symptoms of syphilis after thorough treatment ending four years before. His wife and children were healthy, and the Wassermann test was negative. But on general principles he thought he had better have a prophylactic injection of salvarsan, and an intra-venous injection of 0·5 grm. was given him by a skilled and experienced Berlin specialist. Nausea, vomiting and diarrhœa at once followed and he died the fourth day in convulsions. In a second case an intra-venous injection of 0·1 and 0·2 grm. in the course of a few days was followed by paralysis of both arms. The patient was an anæmic man of 33. The reaction of degeneration was pronounced, but conditions gradually improved in the course of six months, although the arms are still weak. There was no suspicion of syphilis in this case, and the salvarsan was given merely as a means of administering arsenic in a convenient form to influence the pallor and physical depression which had persisted after an operation for chronic appendicitis. No cause for the anæmia was discovered.—*Journ. A.M.A.*, vol. lxi, p. 520.

* Since writing the paper I removed the entire colon in this patient with a very satisfactory result.

A LECTURE

ON

THE EXAMINATION OF THE UPPER AIR-PASSAGES.*

By HAROLD BARWELL, M.B., F.R.C.S.,
Surgeon for and Lecturer on Diseases of the Throat, St. George's Hospital.

LADIES AND GENTLEMEN,—The title of my lecture this afternoon is the examination of the upper air-passages. After selecting the subject, it has seemed to me that some apology is due to you because of the elementary nature of the subject. Still, although I expect that I shall say very little which you have not already heard, I hope I may be able to make a few points which will stick in your minds, and be of actual practical value afterwards. Indeed, there is no section of our practice in which the method of examination is more important. The orifices of which we shall speak are exceedingly sensitive, and any patient who has been examined on various occasions by different practitioners becomes a very good judge as to whether an examination is done skilfully or not; so that by the method of examination one can gain or lose a considerable amount of prestige. As I have just remarked, these parts are very sensitive, and of course, anything in the way of the least roughness is to be strongly deprecated. But, on the other hand, I would impress upon you that too timid an approach to your patient in these examinations is to be avoided almost as much as undue roughness. Such excessive timidity deprives you of the confidence of the patient, it tickles the throat or other part being examined, and does a good deal of harm. What it is most desirable to cultivate is a gentle yet firm touch, and the skilful production of steady pressure. One generally finds students begin to examine patients by taking the instrument delicately on the tips of their fingers, and holding it at arm's length. The patient gets no confidence in such circumstances; you must be firm in your contacts, and get as much into contact with your patient as possible. In introducing the nasal speculum, for example, one should have as much of one's hands and fingers against the patient's face

* Delivered at the Medical Graduates' College and Polyclinic, London.

as one can; and the same applies to examination of the larynx and the pharynx. When this is secured, if the patient starts or moves at all, your contact informs you of the movements, and enables you to move in sympathy and so to avoid jabbing the instrument against sensitive parts. That is a small point, but a very important one.

Then as to the source of your illumination. That also is an important matter, but I shall mention only one or two points. It is essential, in looking down a long tube, to ensure that the pencil of light coincides exactly with the axis of the tube and also with the line of vision. The electric forehead lamp is a very good method of examination, and this lamp can be carried about to patients' houses, but it is of no use for the purpose of looking into the deeper parts of the nose, or down the trachea, because the beam of light does not coincide with the line of vision and thus a shadow is cast. You must have a forehead mirror with a central aperture, through which the eye looks. It requires some practice to use a forehead mirror. You should practise throwing the beam of light on to any desired object, and be able to see the illuminated area at once. A good deal of time is lost in trying to get this adjustment. Many beginners are found not to be using the right eye over which the mirror is placed, and the result is confusion and a headache. With regard to the light, the brighter the light, the better it is. It is impossible to get a good view of a difficult object, such as the naso-pharynx, unless there is a very good light. You can get a brighter light if you have a lens in front, but it is then harder to use; you must throw the light on to the mirror, and a very slight movement throws the mirror out of the illuminated area. For the best illumination I use a big Nernst lamp behind a lens.

We will begin with the nose. Before introducing a speculum into the nose, one should examine its exterior. One can gain much information by such outside inspection, by noting its shape, its narrowness, or the collapse of the alæ nasi. In many cases of nasal obstruction, the alæ nasi are badly developed and slit-like; on inspiration, they are drawn in like valves and are a fruitful cause of nasal obstruction. If you do not examine for this before you use the speculum, you are not likely to detect it. Also any such condition as dislocation of the anterior end of the triangular cartilage is likely to be missed if you do not examine the nose before introducing the nasal speculum, because the dislocation is so far forward that the speculum will hide it. Or there may be a dislocation of the inner limb of the lower lateral cartilage, which makes a small projection further forwards than the anterior end of the triangular cartilage, and softer and more movable. One should test the patient's respiration, making him breathe quietly through the nose, without sniffing; then occlude one nostril, and then the other, alternately. For this purpose, never push against the outside of the nose; this simply pushes the whole organ over, and alters the shape of the other nostril. Instead, place the ball of the thumb over the nostril which you wish to close. Next, one comes to the introduction of the speculum. The type of speculum is important. I am only mentioning my own opinions on the subject, because in regard to the use of instruments, as in regard to other things, everyone has his own pet ideas; and an instrument which seems to suit one examiner very well does not suit another. There are, however, certain points about them which are worth mentioning. A speculum, I think, should have the handle directed upwards, so that on introducing the speculum you lift the tip of the nose and can see along the floor. The channel of the nostrils is directed upwards, and you cannot see along the floor of the nose unless the tip is tilted upwards. You can use either Thudichum's or Lennox Browne's speculum, which has a slide instead of a spring. With both of these you can tilt the nostril and get a good view along the floor; and having no downwardly directed handle, like Fraenkel's or Killian's, it does not get into the way of instruments. If you use a Thudichum's speculum, you should see that the spring is a very light one, or there will be much discomfort to the patient from forcible dilatation of the nostril. Introduce it first in an upward direction, and, when it is in place, tilt it so as to bring the leaves horizontal, and then you can see along the floor of the nose. The person being examined should sit facing you in the ordinary posture, and you should sit with your head on the level of the patient's. The natural tendency of the patient is to throw the head back, and then you see only towards the roof, and not along the floor of the nose at all. The speculum should be

compressed between the first and second finger, with the thumb in front, then the fourth and fifth fingers are free to rest on the face and the bridge of the nose, and this will enable you to move the patient's head up and down or from side to side as desired.

On looking into the naris you see the inferior turbinal, a round red body, and if it is not too large, you should be able to see, to some extent, along the floor of the nose under its concavity. If the turbinal is enlarged and causing obstruction you will see it pressing on the septum. But in many people who suffer from nasal obstruction due to enlargement or to a puffy condition of the turbinals, these bodies are found to be shrunken at the time of the examination, chiefly as the result of the nerve-stimulation which the fear of examination has produced. You can sometimes see the turbinals actually shrink during examination, and hence the turbinal body may appear of normal size, although in the ordinary condition of the patient it causes obstruction. But you will see a shallow groove in the septum, bounded in front by a small semicircular ridge, which corresponds to the anterior margin of the inferior turbinal. That shows that the turbinal is the cause of the obstruction. In others you will see strings of mucus stretching between the surface and the septum, which could only be there if they have been in contact. Above the inferior turbinal one sees the middle turbinal. It should be paler than the inferior turbinal, and you can see more along its lower surface. Under the middle turbinal you see the middle meatus, and this is a very important region from the point of view of disease of the accessory sinuses. Under the middle turbinal—that is to say, if it is not too swollen—you can see a sharp semicircular ridge bounding a groove; this is the hiatus semilunaris, bounded by the processus unciformis. Into the hiatus semilunaris the frontal sinus, the antrum and the anterior ethmoidal cells open, so it is in this position that you see pus coming into the nose if there is suppuration of any of these sinuses. In many patients with rhinitis—general superficial inflammation of the nose—you will see muco-pus scattered about the naris, but if there is a stream of creamy pus running from a localised spot in the nose, it is evidence that there is disease of an accessory sinus. In rhinitis there is muco-pus, but foreign bodies and necrosis are the only other conditions which produce true suppuration in the nose. In children a unilateral discharge of pus is generally due to a foreign body. In adults, such a discharge of pus is nearly always due to disease of an accessory sinus. A localised stream of pus in the nose, therefore, is strong evidence of disease of an accessory sinus. You must then track it to its source, and discover the sinus from which it proceeds. If you find pus below and external to the middle turbinal it is certain to be coming from the anterior group of cells—the frontal sinus, the anterior ethmoidal cells or the antrum. You should wipe away the pus and watch for it to reappear. A little pus in one particular place may have been blown into it, but if you wipe it away and watch it reappear, and it does so in a few minutes, it must proceed from a sinus in the neigbourhood. If the pus appears between the septum and middle turbinal, it must be coming from one of the posterior cells, the posterior ethmoidal cells or the sphenoidal sinus.

There are many other methods of examination which will now help you. If one of the anterior group is affected, the first thing is to diagnose, or exclude suppuration in the antrum, which is the most commonly affected sinus. And the most straightforward way to do this is to tap the antrum and see if it contains pus. You can do this with a trocar and cannula in the inferior meatus, as high up and as far back under the attachment of the inferior turbinal as possible. It is not painful if you first apply 20 per cent. cocaine to the wall. If you wash out the antrum you may syringe pus from other parts of the nose. It is better to use a syringe which fits the cannula, and inject some sterilised water, about one ounce, and immediately suck it out again into the syringe. This method fills the antrum with fluid, and you can suck some back; and if there is pus in the antrum, it will always come out in that way. If you do not find pus in the antrum, and the pus still comes down under the middle turbinal, it is probable that the case is one of frontal sinus trouble. I do not propose to go further into detail with regard to this.

The next accessory method of diagnosing the presence of pus in the sinuses is that of transillumination. For the antrum, you put a bright electric light, shielded by a glass, into the mouth of the patient when he is in a dark room, or his head and that of the observer are covered by a dark cloth.

When transilluminating the antrum, the light passes through the cheeks, and lights the circum-oral region with a glow. It also passes up through the antrum and through its roof, illuminating the soft parts of the orbit. One thus sees a crescentic area of light, corresponding to the infraorbital margin. That is the antral *tache*; and if the antrum is suppurating, the antral *tache* is absent. In addition to this *tache*, if you pull down the lower lid you will find the sclerotic brightly illuminated, and that is a more delicate test. And generally you can obtain the pupil reflex from the retina, which is slightly illuminated. Those three are absent in most cases of antral empyema. There are two additional *taches* of light. One is the lachrymal *tache*, below the inner canthus, which passes through the lachrymal bone, but that light may come through the nose or through the antrum. If you plug the nose or the antrum the light may still be present, but if you plug the nose and the antrum, that light is always absent. A third little glow of light is over the bridge of the nose, corresponding to the nasal bones, and that is known as the nasal *tache*. If much ethmoid has been cleared out, it is often very bright, but it comes through the nose. I mention these because there may be some confusion if you do not know them. Transillumination of the frontal sinus is done by putting a vulcanite funnel over the end of the lamp and pressing it as far back along the floor of the frontal sinus as possible, but in people with shallow orbits the method is unsatisfactory. In people with deep orbits it is more reliable. If the frontal sinus lights up, you get a glow of light over the forehead corresponding to the size of the sinus, with a sharp margin, showing a scolloped edge, such as the frontal sinus naturally has. If you get it definitely, that is easily recognised, and shows that the frontal sinus is not suppurating. On putting the lighted funnel on to the margin of the orbit, you will always get a considerable glow round it, due to the diffusion of light through the soft parts, just as if you were to put it against the palm of your hand. And that is liable to cause confusion, too, but it is not the same as the frontal *tache*, and it has not got a sharply defined edge. Transillumination is a useful secondary aid. It is unusual to get so much asymmetry of the antra that one will light up quite clearly and the other be quite dark without there being disease present. In many patients the bones are so thick that both antra are quite dark. It may be due to double empyema of the antrum, or it may be because all the bones are thick. If one side lights up clearly, and the other side is dark, it is possible that there is antral suppuration, but do not diagnose it simply because you find darkness on transillumination, but only if you find pus present as well. It is not a sufficient ground of diagnosis to find simply one antrum dark and the other light. Darkness on one side may be due to the fact that the patient has had antral suppuration years ago, and there is permanent thickening of the lining. But if you find pus in the middle meatus and that corresponding side is quite dark and the other side light, it is strong presumptive evidence of antral empyema. If there is pus in the middle meatus, and you know that either the antrum or the frontal sinus is affected and you want to exclude one or the other, transillumination is useful. For example, if you have diagnosed antral empyema, and you want to see whether the frontal sinus is affected as well, transillumination is very valuable. If there is a good frontal *tache*, the frontal sinus is not affected, and you are dealing with a case of antral empyema alone. This darkness on transillumination is not due to the presence of pus, because if you wash out the antrum and transilluminate, the antrum is still dark; it is due to hyperæmia of the lining membrane, and it only slowly clears up after the disease gets well. Sometimes it never clears up, in spite of the antrum healing, so that one side is dark and the other light, although there is no suppuration. In the case of the frontal sinus the method is still less useful. If you get a really good and clear frontal sinus *tache*, you may say there is no suppuration present there; but if it is dark, it does not prove the presence of pus at all. The frontal sinuses are so commonly asymmetrical that the fact that one side is seen light and the other side dark is not so useful as in the antum. For diagnosis of disease in this sinus there is an additional method, namely skiagraphy; it is a good accessory method. There is no doubt that a really skillful radiographer, who will take a good photograph of the skull, can help you to a great extent in telling whether pus is present or not. Especially if the disease is confined to one side, the affected sinus gives much less transillumination than does the normal sinus, and its margins are distinctly blurred. That applies

also to the antrum, and to a limited extent to the sphenoidal sinus. It is a very important matter to have a skiagram of the frontal sinuses before one operates on them, as it enables one to see the exact size and shape of the sinuses, and whether any diverticula are present. Skiagraphy is also useful in conjunction with transillumination. For instance, if you suspect the right frontal sinus to be the seat of disease, and it is dark on transillumination and the left one is clear and large, it is possible that the right frontal sinus is smaller, or absent. If you find by skiagraphy that it is of ample size, and yet does not transilluminate while the other does, there is good evidence that that dark frontal sinus is opaque because it is suppurating.

I need not describe the appearance of the fauces, because it is so familiar. In examining, I prefer the short tongue depressor of Lack's pattern, because this instrument enables one to get into contact with one's patient. You hold it between the first two fingers and thumb, and then you can put your last two fingers under the patient's chin and regulate the pressure which you make on the tongue. If the patient moves during the examination your whole hand moves with him, and you can get a good view without causing him to retch.

There is a method of examination which may be worth mentioning to enable you to look into the tonsils and the supra-tonsillar fossa—a method introduced by Killian. It is to put the patient more sideways, introduce the tongue depressor, and use another tongue depressor to retract the cheek; one can then get a more direct view of the lateral parts of the pharynx.

In looking at the naso-pharynx, which is the most difficult of all these examinations, I, personally, prefer to use a straight mirror, 10 mm. in diameter, for most patients, with a straight handle like a laryngoscope. The whole secret of success in looking at the naso-pharynx is the right use of the tongue depressor. It should be introduced just far enough to depress the tongue sufficiently, and not too far, otherwise the patient will retch. You should then slide the mirror between the right faucial pillar and the uvula, but without touching either. You then catch sight of the back of the nasal septum, and on following that up by slightly raising the handle, you can very quickly see whether adenoids are present or not. The septal column expands above into a large concavity like a vaulted arch; but if adenoids are present, this is occupied by a convex mass. The adenoid looks smaller than it is, owing to the effect of foreshortening. It is harder to examine the choanæ than to see adenoids, because you must depress the handle in order to get the mirror into a more vertical position. If the patient raises the palate on opening the mouth, instruct him to breathe in gently through the nose; in that way you can generally get the palate down. Unless the patient raises the palate, it is better not to give any instructions about breathing, because breathing will be done naturally without instructions. If the throat is very sensitive, and the patient retches on slight provocation, it may be necessary to apply a little cocaine. It is better not to spray it on the throat, because the impact of the spray is sufficient to cause retching, and once the patient has begun to retch the examination is rendered much more difficult. It is wiser to apply it on a cotton-wool swab to the base of the tongue, because both in this examination and in that of the larynx it is the region of the base of the tongue which initiates the retching reflex. Mere holding out the tongue is sometimes sufficient to cause retching. It is very important to get a good view of the naso-pharynx in cases where there is obstinate obstruction to the Eustachian tube, not only because you may find some fairly trivial lesion which you can treat, but because you may thus detect a serious malignant growth in the naso-pharynx. The only early symptom of the ordinary malignant growth in the naso-pharynx, endothelioma, is Eustachian obstruction. In the case of patients who have persistent Eustachian trouble which does not clear up fairly readily under treatment, there should be a thorough examination of the naso-pharynx. I saw yesterday a patient who had been treated for Eustachian obstruction twelve months ago, and who has now got inoperable malignant disease of the naso-pharynx.

I show you Holmes's pharyngoscope, an improvement on the earlier pharyngoscopes as it is passed through the nose, and there are few patients on whom it cannot be passed comfortably, provided a little cocaine be used. If one side of the nose is narrow, you can pass it on the other. It is possible to see the back part of the nose and sometimes the sphenoidal orifice; it gives a clear view of the naso-pharynx, and is a valuable instrument as a stand-by for those cases in which you cannot see this region with the post-nasal mirror.

You know the ordinary way of looking at the

larynx. You should sit a little higher than the patient, let the patient's body lean a little forward and have the head thrown somewhat back, then you can get a view towards the anterior commissure and the anterior ends of the cords. That is the most difficult part to see, and it is the most important part to see in many cases, because it is from the junction of the anterior and middle third of the glottis that most benign growths spring; and it is essential to see that region if the patient complains of hoarseness. Hold the tongue out with a cloth, in this way—with the thumb on the top of the tongue, and the second finger underneath. That gives you the index finger free to place on the teeth, and to lift the upper lip and moustache if it is in the way. You can in this way get good contact with the patient, and can move his head backwards or forwards as required. Do not pull the patient's tongue out forcibly; tell him to protrude it, and when he does so, hold it out. Do not drag the tongue downwards, or you may hurt it against the lower teeth; and the more you drag the tip down, the more will the dorsum arch upwards. Thirdly, pass the mirror so that it disappears, or "sets," behind the arch of the dorsum of the tongue, and then lift it backwards and upwards, so as to raise the soft palate. For a good view it is essential that you should put your mirror right against the posterior pharyngeal wall, not with a light tickling movement, but with a steady, firm pressure, and then the patient will not retch. It is easier than examination of the naso-pharynx. Tell the patient to breathe deeply, but quietly, through the mouth. If you can persuade him to take a deep breath directly he feels a tendency to retch, that tendency will disappear.

The first difficulty in the examination of the glottis is the presence of a dependent epiglottis. In many cases it is the incorrect position of patient and mirror which makes the epiglottis appear dependent; but in some cases the epiglottis does hang over the larynx like a lid. You can often raise the epiglottis by telling the patient to phonate sharply on the sound "*eh*," rather than "*ah*," and on a high note, or even get him to go up the scale in a staccato manner. Another method is to make the patient phonate until he is out of breath, and then tell him suddenly to take a deep breath. That gasp opens the whole larynx, and you will have a good view. If all these devices fail, you will have to use the epiglottis tractor. It is troublesome to use, and it is necessary to apply a good deal of cocaine along the base of the tongue; still, in important cases it may be quite necessary. Here I show you Escat's epiglottis tractor. It is like a tongue depressor, but with two little balls on the curved end. You pass the mirror with the right hand, and the epiglottis tractor with the left. It is not put over the epiglottis, but in front of it into the glosso-epiglottic fossa. A downward and forward pressure with these knobs on the base of the tongue will lift up the epiglottis. A method which is useful occasionally when you have to do operative procedures on the larynx, such as removing papillomata from the vocal cord, when there is a very dependent epiglottis, is to pass a suture through the epiglottis. The instrument suggested by Horsford for this purpose is a small forceps which passes a little needle, and catches it on the other side. You can hang a metal clip on to the end of the thread, and so pull the epiglottis forward. It is quite a painless procedure, it does no harm, and it enables you to get a good view when such a view is important. As a rule, that is not necessary for ordinary examination. There is a little dodge of Killian's which will enable you to get a good view of the posterior wall of the larynx, and often enables one to see directly along the trachea to the bifurcation. In this procedure, instead of the patient sitting down he stands up, and bends the head forward at the neck, while the observer sits in a chair and looks upwards to the patient. The tongue is held and the mirror is passed in the usual way.

I need not allude at length to the direct method of examination, because I think you are probably all familiar with it, and time does not permit me to enter into it fully now. It is very useful for certain cases, but I do not think direct examination should be used in preference to indirect for most manipulations in the larynx in adults, such as for removal of papillomata from the vocal cord or cauterising a tuberculous ulcer. The indirect method, although it requires more skill, is far less unpleasant to the patient. But the direct method is absolutely invaluable for many cases, for any manipulation below the epiglottis or in the trachea, for all work in the œsophagus, which has been opened up to our view by the direct method, and especially for laryngeal work in children. It is an enormous advance to be able to remove multiple papillomata in children safely and certainly at any age by this direct method. In the old days when only the indirect method was available, the only thing one could do in the case of small children with these papillomata was to give them chloroform to the full extent, in the sitting posture, in addition to cocainising the throat, and then to remove the growths with forceps guided by the mirror. This was a very dangerous method, as in addition to the danger of deep chloroform anæsthesia in the upright position, there was the risk of the entrance of blood and fragments of the growth into the lungs. But the greatest advantage of this addition to our resources lies in the treatment of foreign bodies in the trachea, bronchi and œsophagus, and many patients have now been saved by the use of the direct method who would otherwise most certainly have died.

September 22nd, 1913.

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NEPHROPTOSIS: ITS CLINICAL SIGNIFICANCE.

By WILLIAM BILLINGTON, M.B., M.S.Lond., F.R.C.S.,

Surgeon to the Queen's Hospital, Birmingham.

DISPLACEMENTS of abdominal viscera have received serious attention only within recent years, and even now do not receive sufficient consideration from the profession. Ptosis of one or more abdominal organs is very common, and is a penalty paid by the human race for the upright posture. Glénard drew attention to the serious symptoms associated with general visceroptosis, but while true Glénard's disease is rarely met with, examples of prolapse of single organs, and especially of the kidneys, are very common. A wide clinical and operative experience of renal mobility has convinced me that it is a potent cause of ill-health, and is responsible for a serious fall in the working efficiency of many otherwise healthy people. I will not now enter into the many problems connected with general visceroptosis, excepting in so far as it is necessary to define the relation between nephroptosis and ptosis of other organs.

Movable kidney and enteroptosis.—Cases of general visceroptosis have been usefully divided into two classes—congenital and acquired. Congenital visceroptosis, whether the kidneys be involved or not, falls outside the scope of this article. It is due to defective development and cannot be remedied by surgical measures. Treatment must be on general lines, must begin early, and must be continued during the whole period of evolution from childhood to adult life.

Acquired visceroptosis may involve one or many abdominal organs. General ptosis of the abdominal viscera, including the kidneys, associated with severe neurasthenic symptoms and local kidney disturbances, such as Dietl's crises (acute attacks

of renal colic caused by torsion of the renal pedicle or obstruction of the ureter), is termed Glénard's disease. It is not common; I do not see more than two or three cases in the course of a year. Far more common is the form of visceroptosis seen in women who have borne many children and whose abdominal walls are lax and pendulous. In the erect posture the bowels fall into the lower abdomen, and practically every organ below the diaphragm assumes a lower level than normal. This form of visceroptosis is so common that if estimated by some undue prominence below the umbilicus is almost universal in women over thirty. The symptoms are rarely severe, and are easily relieved by suitable external supports and correction of errors of dress.

In a third and very important group of cases there is not much enteroptosis, and yet the symptoms are so severe as to cause chronic invalidism. The patients are for the most part unmarried, and if married have had fewer children than is the average. They have at one time been normal; are comparatively young when the symptoms begin; are organically sound, and usually have a good family history. Rest alone seems to benefit them, but only so long as they are resting. In a majority of cases examination reveals mobility of one or both kidneys.

In dealing with visceroptosis a distinction must be made between the kidneys and the intra-abdominal viscera. The kidneys are normally outside the peritoneum, and lie in special sockets at the sides of the vertebral column. Developmentally and anatomically they are not abdominal organs. Enteroptosis and nephroptosis are separate conditions although subject to the same laws. They are usually associated to some extent, but only in Glénard's disease, which is comparatively rare, is the association complete. Usually, either nephroptosis and its symptoms are predominant, and the associated enteroptosis is of little account; or the enteroptosis is the principal trouble and the nephroptosis of little account. This distinction is important from the point of view of treatment. When nephroptosis is predominant external support of the abdomen is unsatisfactory, and nephropexy is indicated. For enteroptosis, corsets, etc., give good results, and nephropexy should not be performed.

Definition of nephroptosis.—There seems to be a good deal of confusion as to what constitutes abnormal mobility of a kidney. The normal kidney moves with respiration, the range being about half an inch. This movement is caused by the excursions of the diaphragm, and is common to all the sub-diaphragmatic viscera. I would define a pathologically movable kidney as one the position of which is affected by influences other than respiration and in particular by gravity. This is possible only when there is abnormal laxity of the ordinary kidney supports.

Tests of abnormal mobility.—I am accustomed to apply two tests to palpable kidneys to determine whether they are pathologically mobile. First, does the kidney assume a lower level when the patient is standing up than when she is lying down, *i.e.* is its level influenced by gravity? Second, can the kidney be pushed to a higher level by the examining hand, and can it be prevented from ascending during expiration by grasping its lower pole? Even when the normal kidney is exposed from the loin it can neither be pushed up nor its respiratory movements restrained until it has been enucleated from its bed.

Ætiology.

Heredity is a factor of some importance. Nephroptosis is often found in several members of the same family, the renal mobility being predisposed to by abnormal laxity of the natural supports of the kidneys. The following are the more important exciting causes:

(1) *Trauma.*—Careful investigation of a large number of cases leads me to assert that muscular overstrain is the most important direct cause. In men, a history of severe strain or injury to the back is almost always forthcoming. In women the relation is usually less easily established, but the patient's habits and occupation and the absence of any other satisfactory explanation frequently indicate that the kidney was started on its downward path by a strain or direct injury to the loin. Trauma is particularly liable to cause displacement of the kidneys in girls at and soon after puberty. As the pelvis assumes the adult female shape changes occur in the lower part of the renal fossa. It becomes wider and shallower and affords less resistance to descent of the kidneys. At this time violent exertion, especially in girls whose general muscular tone is below normal, is apt to

strain the kidney supports and initiate nephroptosis. I am certain that the modern tendency for growing girls to take part in active games such as hockey, tennis, etc., is responsible for many loose kidneys.

(2) *Errors of dress.*—Constriction of the waist by corsets and bands had been generally held to be an important cause of nephroptosis. That such constriction does cause enteroptosis and displacements of the pelvic organs is probable, but that it is the primary cause of nephroptosis is very doubtful. The waist-line is *below* the level of the normally placed kidneys, and constriction of the waist rather tends to support than to force down these organs. If, however, the kidneys have fallen below the waist-level, such constriction tends to aggravate the nephroptosis.

(3) *Child-bearing.*—After confinement, while the abdominal muscles are lax, any unusual strain is more likely than usual to induce prolapse of the kidneys and intra-abdominal organs. The result is general visceroptosis in which the kidneys share to a greater or less extent, rather than nephroptosis alone. The great majority of my patients who suffered from the more severe symptoms of nephroptosis were either unmarried or had borne only one or two children.

(4) *Constipation.*—The drag of a loaded cæcum and colon tends to loosen the peritoneal attachments at the hepatic and splenic flexures, but constipation is so much more common than nephroptosis that its influence in causing the latter is difficult to estimate. Further, constipation is a marked sequel of renal mobility and it is easy to mistake effect for cause.

(5) *Changes in and around the kidney.*—Diminution of the capacity of the renal fossæ, *e.g.* by enlargement of the liver, scoliosis, etc., and increase in the size of the kidney itself from hydronephrosis, cystic disease, etc., tends to bring about displacement of the latter. Absorption of the perinephric fat in wasting diseases has been given as a cause of nephroptosis. I fail to find any justification for this theory. The fat around the kidney has very little supporting function and acts merely as packing, protecting the kidneys from injury. Diminution of this fat is one of the first *results* of abnormal mobility, its place being taken by connective-tissue adhesions between the capsule of the falling kidney and surrounding structures. These adhesions are, as it were, thrown out to check further descent. Further, there is no evidence to show that nephroptosis is at all common in such severe wasting diseases as malignant cachexia and advanced phthisis.

Method of Physical Examination.

Loose kidneys are often overlooked because they are not specially sought for. They easily escape detection during a routine examination of the abdomen with the patient recumbent, for then even freely movable kidneys occupy a more or less normal position. Examination may be difficult on account of the patient's nervousness or obesity, but most of the failures are due to defective methods. During recumbency it may be difficult to dislodge a loose kidney from under the ribs, especially on the left side. The hollow stomach acts as an air cushion between the diaphragm and the kidney, and minimises the downward thrust of the former during deep breathing and coughing. Even competent observers often fail to recognise a freely mobile left kidney in the recumbent position.

To obtain full information respecting a possibly mobile kidney, the patient must be examined both standing up and lying down. Neither position is altogether satisfactory, but the disadvantages of one are to a large extent compensated for by the other. In the erect position the extent of the displacement can be better estimated, and kidneys can often be felt which are not palpable during recumbency. On the other hand, it is more difficult to obtain relaxation of the abdominal muscles, and less easy to estimate variations in the size of the kidney.

Examination in the recumbent position.—The abdomen is completely uncovered and all tight garments loosened. The patient lies on a firm, roomy couch of convenient height and ample length. Both sides of the couch should be equally accessible to the examiner. The shoulders and upper part of the trunk are raised on an inclined plane, and a small pillow is placed under the head (Fig. 1). If the patient is nervous, or the muscles rigid, a second pillow may be placed under the knees in order to allow slight flexion of the thighs without effort on the part of the patient. The examiner sits on the side to be examined. It is impossible satisfactorily to examine both loins from the same side. A general examination of the abdomen is first made with a view to

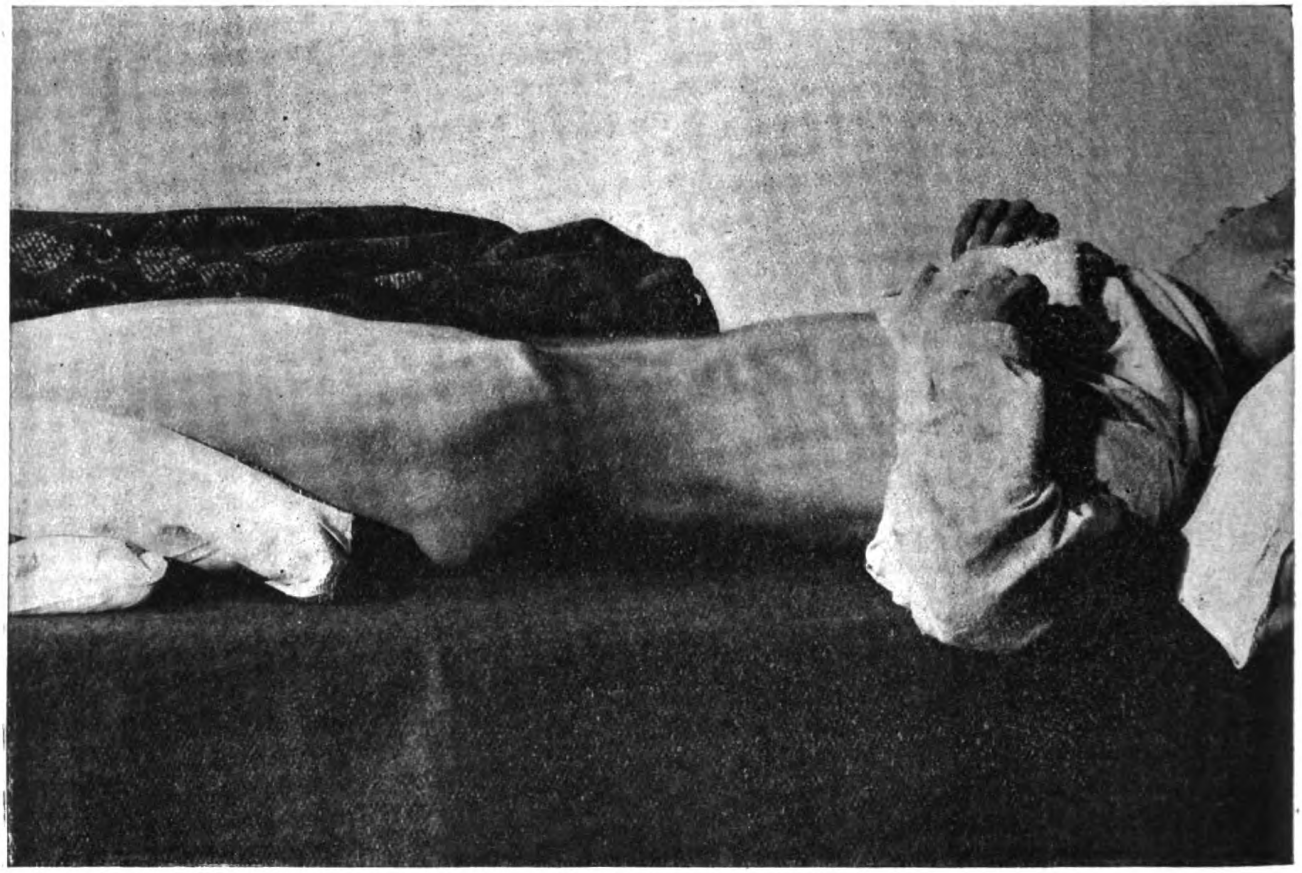

FIG. 1.—Recumbent position.

excluding other abnormal conditions, and at the same time gaining the patient's confidence. Then, if the right kidney is being examined, the loin is enclosed by the left hand in such a way that the thumb occupies the angle between the last rib and the outer edge of the erector spinæ muscle, while the fingers lie in front. The patient is instructed to allow the back to rest comfortably on the couch, and to take a series of long, deep breaths. During the first few no attempt is made to entrap the kidney. When confidence has been obtained and breathing is going on satisfactorily, the loin is firmly grasped between the left thumb and fingers at the end of an inspiration. If the kidney comes below the costal margin its presence is at once detected, and if definitely movable it can be held. If the kidney is not felt, it may be above or below the approximated thumb and fingers. While the loin is still grasped as above, a careful examination of the abdomen below should be made with the right hand, which is free. If the kidney is discovered below, attempts should be made to push it back into the loin, the grasp upon which is relaxed to allow of its return. If this manœuvre can be executed it is proof that the lump felt is the kidney. Finally, if the kidney cannot be felt by the left hand, nor its presence lower down be detected by the right hand, the patient should be instructed to cough, with the object of dislodging it. On the right side this is often successful, but, as already explained, coughing not infrequently fails to dislodge even a very movable kidney, and the condition may easily be overlooked if no examination in the erect position is made.

To examine for the left kidney the surgeon sits on the left side of the patient and grasps the loin with his right hand in the way described (Fig. 2). The left hand is now at liberty to explore the lower abdomen or palpate the imprisoned kidney.

Whilst held by the hand which grasps the loin, the kidney can be carefully examined by the free hand, any variations in size or other abnormalities being noted. This method of examination is easy

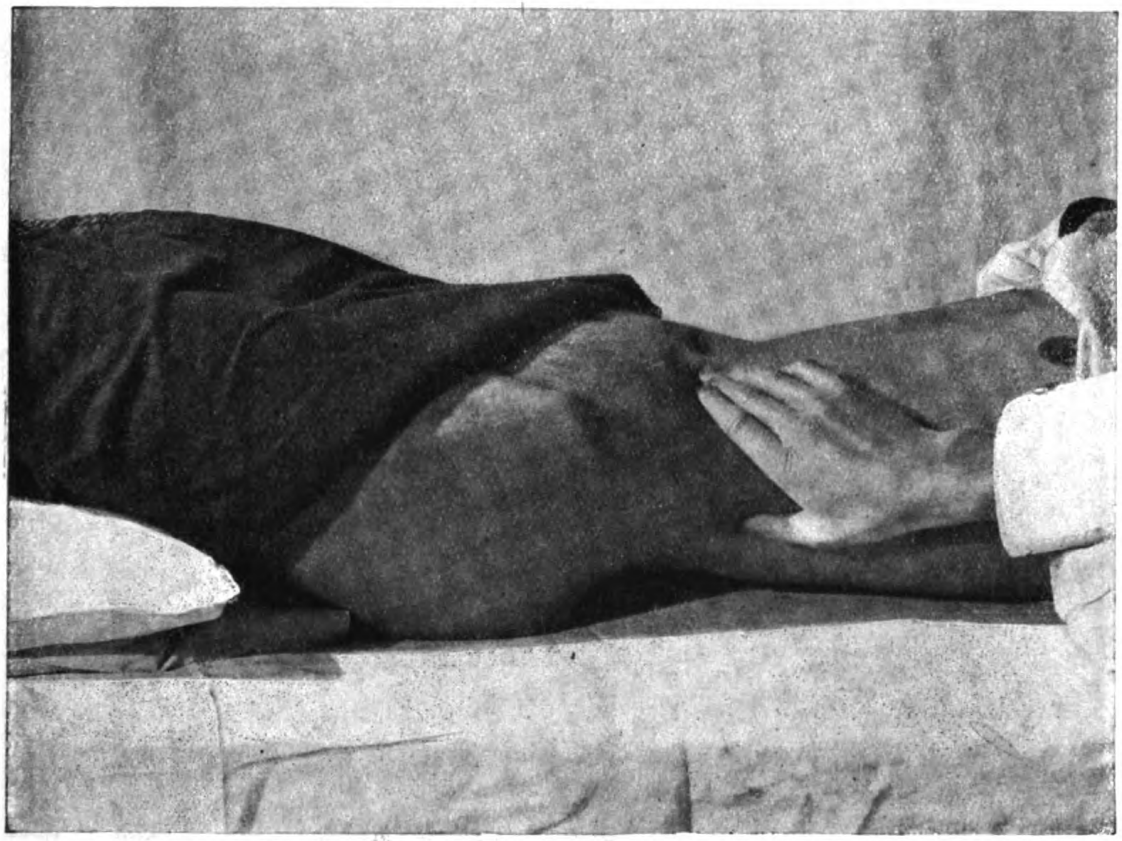

FIG. 2.—Position of right hand in palpation of left loin. The left hand is at liberty.

to carry out, very effective, and does not alarm or incommode the patient. It has several advantages over the usual bimanual method. In the bimanual method novices are apt to compress the loin too forcibly between the fingers of the two hands and so prevent the descent of the kidney. Again, it is more difficult to imprison the kidney and more difficult to differentiate a loose kidney from an enlarged gall-bladder or a Reidel's lobe of the liver, which may also descend between the fingers. Last, and most important, the bimanual method occupies both hands, and the great advantage of a free hand with which to examine the kidney while it is being held by the other is lost.

Examination in the erect position.—The patient's skirts are loosely fastened in such a way that they are suspended from the hips without its being necessary for her to support them with her hands. She is then asked to stand with the body slightly bent forwards and the hands resting on the back of a chair. The examiner stands behind (Fig. 3). For the right loin the right hand is used, the thumb, as before, occupying the angle between the last rib and the erector spinæ muscle, and the fingers lying in front, immediately below the costal margin. To examine the left loin the left hand is similarly placed (Fig. 4). In this way the whole of the loin can be readily palpated, and the kidney, if down, grasped between the thumb and fingers. The level it assumes under the influence of gravity and the effect upon it of respiration can be ascertained. In this position deep breathing and coughing are more likely to bring the kidney within the grasp of the hand than during recumbency. If desired, a loose kidney detected in this way can be held while the patient lies down, and then examined more carefully.

There are one or two signs, which, though not in themselves diagnostic, yet suggest renal mobility. When the kidney is movable the loin has a different feel; it is softer, and gives an impression of emptiness. Again, percussion over the area around the angle between the erector spinæ and last rib, while the patient stands with the body slightly inclined forwards, often elicits a resonant note when the kidney is displaced. These signs

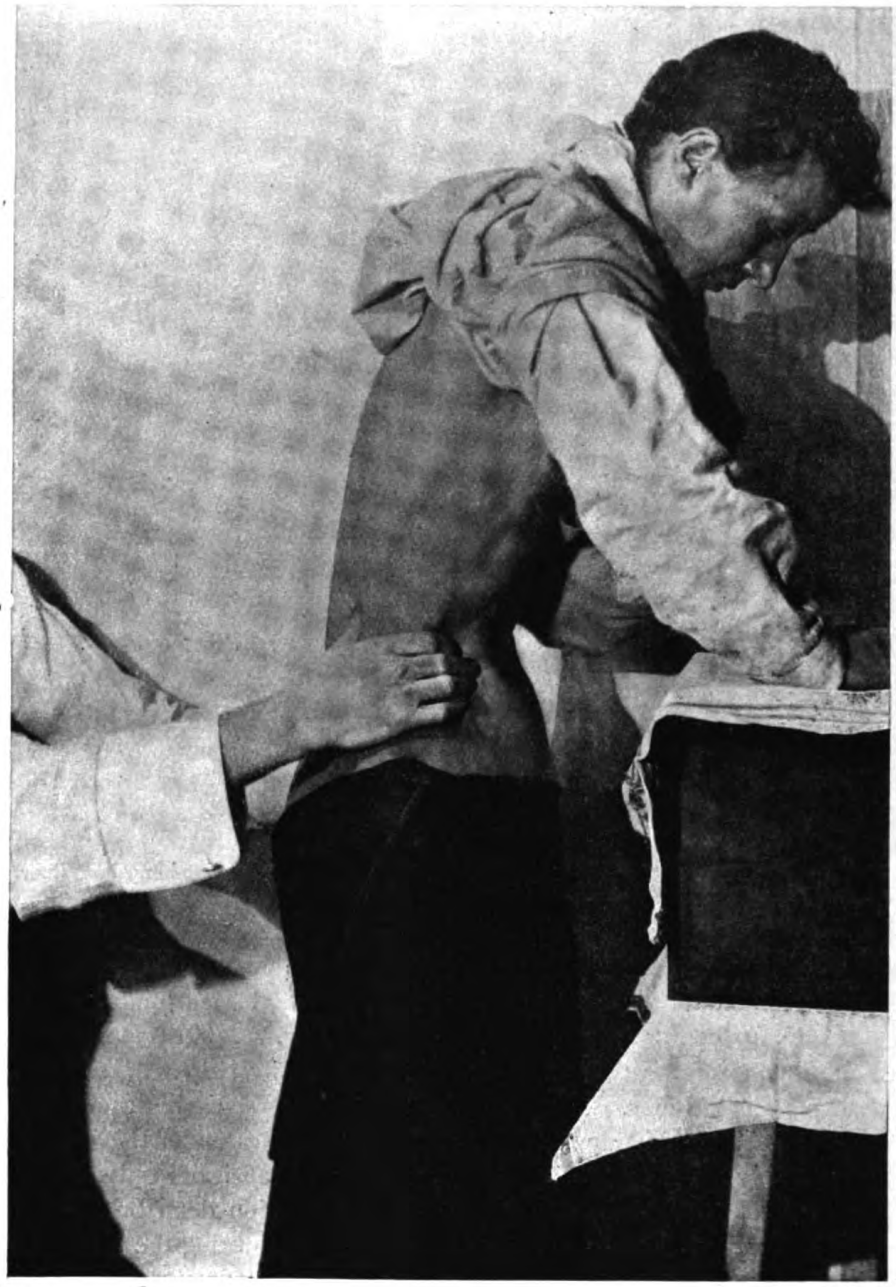

Fig. 3.—Examination of right loin in the erect position.

are especially useful when rotation of the kidney without downward displacement is suspected. It may be impossible to palpate such a kidney in any position of the body, and a diagnosis has to be based on symptoms, the signs just mentioned, and careful examination of the urine for evidence of interference with renal function. Not infrequently such kidneys give rise to albuminuria.

Latterly it has been found possible to demonstrate renal mobility by means of the X rays. Some excellent skiagrams showing the kidneys in their abnormal position have been obtained. At present, however, no reliance can be placed upon a negative result, and in the main the diagnosis must depend upon ordinary methods of examination.

Changes in Kidney and Surrounding Structures.

On opening the loin for the purpose of anchoring a loose kidney, the first thing one notices is a

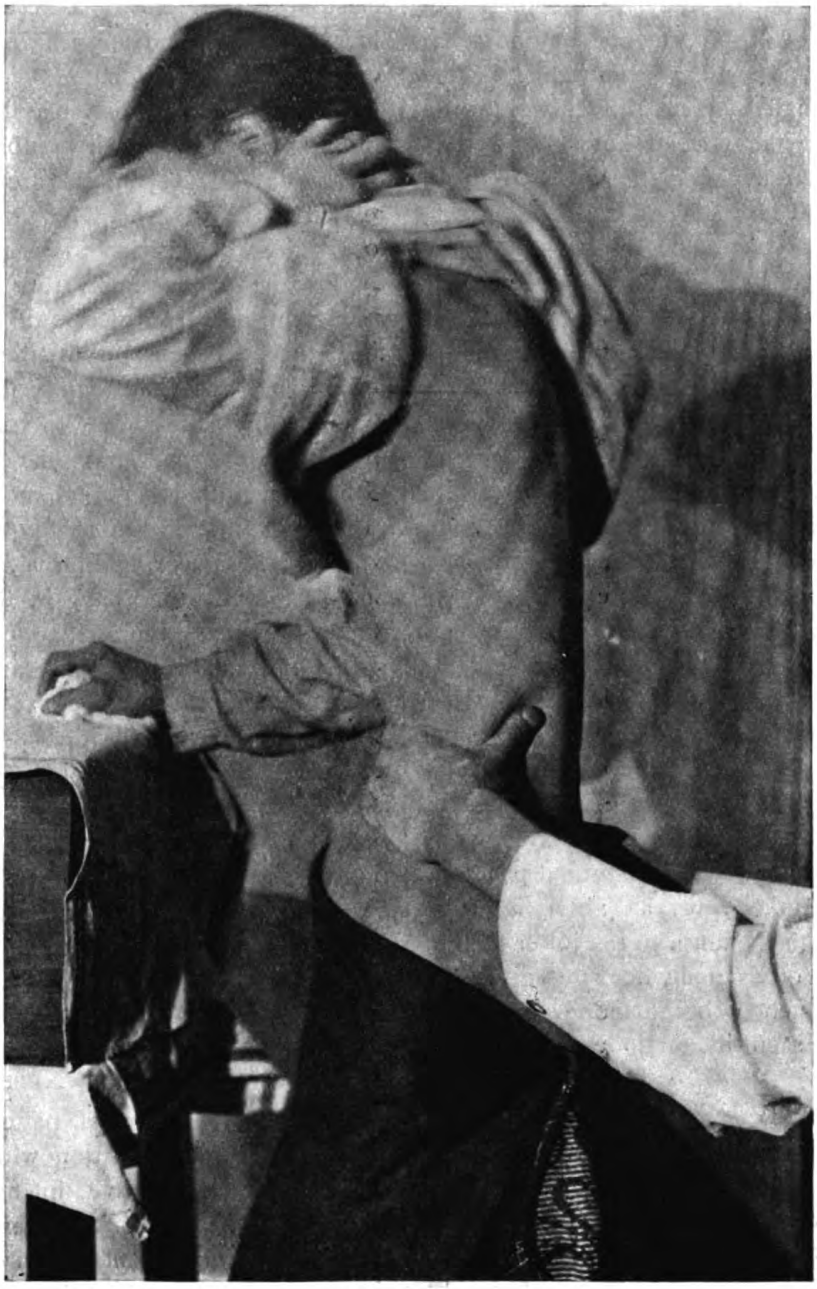

FIG. 4.—Examination of left loin in the erect position.

peculiar emptiness. Instead of the fat bulging into the incision as soon as the lumbar aponeurosis is divided it appears to have fallen away, while the perirenal capsule is much less firmly attached to the muscles of the back than usual. This is noticeable even though prolapse of the perirenal capsule has not taken place with the kidney. The perirenal capsule forms a more definite structure than usual and is easily identified. The peritoneal fold, which usually extends a little way into the loin, may be much deeper than usual and may cover most of the posterior surface of the kidney. Unless care is taken it is in danger of being wounded. The perirenal capsule, instead of being packed with loose granular fat which can readily be separated from the kidney by the finger, is almost empty. Dense sheet-like adhesions connect the fascial capsule with the true kidney capsule. Though loose and open these adhesions are very strong, and it is impracticable to tear them through

with the fingers. Usually they are bloodless, but in cases of recent displacement they may be vascular. If the kidney has from time to time been partially strangled by twisting of its pedicle there may be many large veins among the adhesions. Some of these pierce the true capsule of the kidney and bleed freely on the kidney side when the latter is congested. Clearly they are collaterals produced by periodical obstruction of the renal vein.

A typical movable kidney, as seen during operation, has a characteristic appearance. Unless congested it is smaller and paler than normal and has a curious flabby feel. Its capsule is thick and opaque, and is mottled by opaque, yellowish-white patches of varying size. These patches indicate areas of greater thickness, and to them are attached the denser adhesions. The capsule is rarely very adherent to the renal cortex and usually strips readily even at the site of the denser patches. It is unusual to find the extra-renal portion of the pelvis, or the ureter, dilated, but the intra-renal portion usually is and the calices are more capacious than usual. The papillæ are also flattened. Gross hydronephrosis is rare unless the kidney has become permanently fixed in an abnormal position, or the ureter is kinked over an aberrant artery passing directly from the aorta to the lower pole of the kidney. Only occasionally does the suprarenal capsule remain attached to the kidney and drop with it, but I have seen this on quite ten occasions.

Symptomatology.

The symptoms of nephroptosis are so many and varied that a detailed account of them would occupy more than the whole of the space at my disposal. I can only briefly outline their general character. Apart from those resulting from recent sudden displacements of the kidney or from such complications as torsion of the renal pedicle with partial strangulation of the kidney, temporary complete obstruction of the ureter, etc., the symptoms of renal mobility take the form of chronic functional disturbance. Sometimes one system is mainly affected, sometimes another, but whatever their form they have certain common features. There is almost always more or less neurasthenia and diminution of both mental and physical efficiency. Effort of any kind produces abnormal exhaustion. Rest produces temporary benefit, but speedy relapse follows a return to active life. Travel, exercise and physical exertion of any kind aggravate the condition.

Cases of nephroptosis may be arranged in five groups according to their predominant symptoms.

Group 1.—The symptoms are local and definitely related to the kidney. The principal symptom is local pain, which is due to recent displacement of the kidney, to congestion from obstruction of the renal vein, or to obstruction of the ureter. Sometimes the pain comes on in paroxysms to which the term Dietl's crisis has been applied. These attacks are probably always due to torsion of the renal pedicle and partial strangulation of the kidney. Urinary symptoms are common. Increased frequency of micturition is frequent, especially in women. It is usually diurnal, but may be also nocturnal. Cutting pain in the urethra during micturition may occur. The urine is rarely consistently normal in quantity and quality. Transient albuminuria is common; it occurs when the patient is about and disappears with rest or adequate support of the kidney. Such accidents as ureteric obstruction or twisting of the renal pedicle cause marked diminution of urinary secretion followed by an excessive flow of pale urine. Hæmaturia may follow congestion of the kidney from obstruction of the renal vein. In the chronic cases evidences of renal irritation, *e.g.* renal cells, traces of albumen, etc., are often to be found.

Bacilluria and Nephroptosis.

A large percentage of patients suffering from nephroptosis in association with marked neurasthenic symptoms have infected urine. The organism most commonly present is a member of the *B. coli* group, sometimes in pure culture and sometimes associated with other germs. The explanation appears to be that accidental infection of the urinary tract, probably a common event in women, fails to clear up on account of defective drainage of the renal pelvis from kinking of the ureters. Chronic bacilluria with resulting toxic absorption appears to be responsible for many of the remote symptoms so frequently associated with movable kidney. So long as the kidneys remain movable the ordinary vaccine treatment of the condition is usually unsuccessful. Nephropexy, with the resulting removal of the obstruction to the free escape of urine from the kidneys, is generally

followed by disappearance of the bacilli from the urine, but in a few cases subsequent treatment with an autogenous vaccine is necessary. Bacilluria may exist in the absence of definite urinary symptoms, and can be detected only by bacteriological examination of the urine obtained by catheterisation. Bacilluria constitutes a strong indication for active treatment of the nephroptosis, for its continuance not only perpetuates the ill-health of the patient but has a very harmful effect upon the kidneys themselves.

Group 2.—The symptoms are due to functional disturbances of the sexual organs. In women, these are dysmenorrhœa, amenorrhœa, menorrhagia and metrorrhagia, and persistent leucorrhœa. I have met with examples of each of these conditions for which no satisfactory local condition was discoverable and who have been cured by nephropexy. In every instance the general health was disturbed to a degree unaccountable for by the pelvic symptoms. I would emphasise the necessity for excluding movable kidney before proceeding to operation upon the pelvic organs for symptoms for which no gross local cause is discoverable. Further, the most disappointing patients upon whom I have performed nephropexy have been those whose ovaries had been previously removed, and I have now given up the attempt to restore such persons to the normal state. In men, sexual neurasthenia, nocturnal emissions and spermatorrhœa may be associated with renal mobility and cured by remedying the latter.

Group 3.—Chronic functional disturbances of digestion are common and may be the principal complaint. They are associated with neurasthenia, depression and hypochondriasis. Capricious appetite, flatulence, paroxysmal attacks of vomiting, severe constipation, and occasionally diarrhœa are the usual symptoms. The peculiar feature is that they refuse to yield to ordinary treatment and that no sufficient cause within the digestive system can be found. Dilatation of the stomach and mucous colitis are not uncommon. My results from nephropexy for renal mobility associated with mucous colitis have been excellent, and a permanent cure has followed in practically every instance.

Group 4.—The patients suffer from spinal and cerebral neurasthenia. This is a large group. The leading symptoms are severe and persistent headache, backache, constant "tiredness," periodical fits of black depression, and the "feeling that life is not worth living." Married women cannot do their housework, teachers complain that the strain of standing and teaching causes complete mental and physical prostration, and business men cannot concentrate their minds upon their work, lose their grip of detail, and find that everything, even the writing of a simple letter, requires great effort. The greater the necessity for work the more pronounced the symptoms. These people are irritable, restless and miserable, a nuisance to themselves and all around. They are painfully conscious of their inefficiency, and in spite of treatment and an active desire to get well remain in a state of invalidism for years. They are comparatively young, organically sound, and of good stock. Of them, Weir Mitchell has said: "Why such people should be so hard to cure I cannot say, but the sad fact remains. Iron, acids, travel have for a certain proportion of them no value, or little value, and they remain for years feeble and for ever tired." I would add, for those of them with nephroptosis, that Weir Mitchell's treatment, though of marked temporary benefit, is followed by speedy relapse on returning to ordinary life.

Bearing in mind that in practically every case nephropexy was preceded by prolonged treatment on the usual lines without benefit, my results in this class of cases are most encouraging. Without being able to give the actual figures, I am within the truth in stating that 60 per cent. have been cured in the sense that within a year of the operation they were leading the ordinary life of healthy people.

Group 5.—In this class are placed patients with definite symptoms of mental derangement. Melancholia, suicidal impulses, delusional insanity, and, more rarely, mania may be associated with nephroptosis.

Treatment.

Renal mobility without symptoms does not call for active treatment. There is, however, no guarantee that symptoms will not develop later, and care should be taken to avoid aggravating the condition by errors of dress, violent exercise, etc. Movable kidney associated with local pain, and with sexual, nervous, mental or digestive symptoms, calls for active treatment especially when other measures fail. The treatment is mechanical—*i. e.* by belts, trusses, etc.—or operative. Attempts to effect permanent replacement by posture, prolonged rest or forced feeding are wrong in principle and futile.

Of the mechanical appliances for the support of movable kidneys, Suckling's belt and Treves' truss are the most generally employed. Good results can be obtained by properly fitted corsets. It is, however, difficult to satisfactorily support a loose kidney, for it is deeply placed, and considerable pressure is necessary to overcome the resistance of the abdominal wall. This pressure is always irksome, and if the loin is tender, is insupportable. Further, when the kidney is very movable any sudden effort may force it under the pad, which then lies upon or above it. Belts or trusses are useless if the kidney does not readily return to its normal position when the patient lies down. They are also useless for rotated kidneys. The belt

should always be applied in the recumbent position. A belt may be satisfactory when the degree of mobility is only moderate and the kidney does not fall completely below the ribs. A strong elastic belt with elastic pads of moderate tension is advisable when movable kidney is associated with prolapse of other abdominal organs. Then nephropexy gives only partial relief, and an elastic abdominal belt is necessary afterwards. In some cases it is advisable, before proceeding to nephropexy, to try the effect of mechanically supporting the kidneys. An improvement would be a strong argument in favour of operation should the belt fail to give complete relief or prove too irksome for permanent use.

The only effective way of permanently restoring a loose kidney to its normal position is an open operation. This can now be done with safety, precision, and permanent success. The same rules apply to the selection of cases for nephropexy as for radical cure of hernia. If the symptoms call for treatment of movable kidney the choice between trusses and belts or operation will be determined by (1) the type of displacement, (2) the nature of the symptoms, and (3) the age, occupation, and general condition of the patient. Movable kidneys, however, are less easy to control by mechanical means than are herniæ. Operation is therefore more strongly indicated.

(1) *The type of displacement.*—Freely movable kidneys cannot effectively be controlled by belts, etc. They slip under the pad. Rotated kidneys require operative treatment. The amount of prolapse is usually trifling, and pressure applied externally will not prevent rotation and may easily make matters worse. Operation is required when the kidney has contracted adhesions to surrounding structures, which prevent its ready return into the loin in the recumbent position. Nephropexy is advisable when belts have failed, either because the pressure is unbearable or because they fail to control the mobility.

(2) *The nature of the symptoms.*—In the main, local symptoms definitely caused by the movable kidney call for operation. Tenderness of the loin or kidney make belt treatment impossible. Repeated attacks of acute pain from twisting of the renal pedicle or obstruction of the ureter call for nephropexy. Not only do these accidents cause great suffering, but they may seriously damage and ultimately destroy the kidney. Finally, an enlarged, movable kidney should always be operated upon. If the enlargement is due to hydro-nephrosis, it may be possible to save the kidney by timely operation.

(3) *The personal equation of the patient* is of great importance. The age, occupation and general condition must be carefully considered. Operation is rarely advisable, except to relieve pain, in persons over fifty. In the young, and especially in those who wish to lead an active life, nephropexy is preferable to treatment by belts. Renal mobility undoubtedly diminishes working efficiency, and is a serious handicap for those who have to work. The co-existence of serious organic disease contra-indicates operation, and careful search for such must be made in every case.

Space does not admit of a detailed description of the operative treatment of nephroptosis. An account of the operation devised by me was published in the 'British Medical Journal,' November 30th, 1907, and subsequently in a monograph ('Movable Kidney; its Ætiology, Symptoms, and Treatment,' Cassell & Co., 1910). Suffice it here to say that nephropexy can now be performed with the safety, precision, and permanently satisfactory results that characterise the operative treatment of hernia. Personally, I have operated on 472 patients, and have performed nephropexy upwards of 700 times, both kidneys having been operated on at the same time on many occasions. In this series there have been four deaths—a mortality of less than 1 per cent. To my knowledge in no single instance has the operation failed to permanently anchor the kidney, and on only one occasion has a subsequent operation been necessary. This was for a small hernia which developed in the scar as a result of a severe strain a few weeks after nephropexy had been performed. This is the only instance of weakness of the scar that has occurred. There have been no cases of persistent sinus or permanent pain in the back after operation. These results compare favourably with those obtained in any other branch of operative surgery, and prove conclusively that there are no valid surgical reasons why movable kidneys should not be treated by operative means.

In conclusion, I wish to emphasise the fact that nephropexy does not cure the patient except when performed for purely local symptoms; it only makes it possible for recovery to take place. A patient operated on for renal mobility associated with neurasthenia is still neurasthenic, after the renal mobility has been cured. The neurasthenia must be treated for the usual time. Neglect to appreciate this fact is responsible for many so-called failures and for much unfair criticism. My contention is not that nephropexy cures neurasthenia, but that neurasthenia which was incurable before the operation becomes curable afterwards. To effect the cure, however, operation must be followed by the careful and prolonged treatment necessary for neurasthenia in general. For this reason the patients whose after-progress is most unsatisfactory are those of the hospital class. The necessity for an early return to work and the influence of an unfavourable environment cause delay in the disappearance of neurasthenic symptoms, and a prolongation of the interval between operation and restoration to health.

September 29th, 1913.

GLYCOSURIA.

By W. LANGDON BROWN, M.D., F.R.C.P.,
Assistant Physician to St. Bartholomew's Hospital and Physician to the Metropolitan Hospital.

Of recent years our conceptions of the pathology and treatment of glycosuria have undergone some important modifications, to which I should like to call attention. I do not propose to touch on the excretion of other reducing substances than sugar, or of other sugars than dextrose.

If equal parts of hot urine and hot Fehling's solution are mixed and yield an orange precipitate *without reboiling*, it is almost conclusive evidence of the presence of dextrose or lævulose (Garrod). If in addition the urine is dextro-rotatory, ferments, and yields osazone crystals with phenyl hydrazine which melt at 206° C., the presence of dextrose is certain.

It is physiological for temporary glycosuria to occur if 150-200 grm. of dextrose are taken at one time, but glycosuria is not physiological after any quantity of starch. In Naunyn's phrase a patient who passes sugar after the free taking of starch is virtually a diabetic.

Even in patients who do not ordinarily pass sugar there may be a lowered tolerance for carbohydrates. This lowered tolerance may precede the glycosuria and outlast it. It indicates that the metabolic defect that caused the glycosuria is still present, and that the sugar may return on slight provocation. In all but one form of glycosuria there is excess of sugar in the blood—hyperglycæmia. This may also precede and outlast the glycosuria and has a similar significance to lowered sugar tolerance. The estimation of sugar tolerance and the sugar content of the blood is a valuable guide in estimating the reality of an apparent recovery from glycosuria.

It will systematise our conceptions of diabetes to consider the ways in which glycosuria may be experimentally produced and then the clinical equivalent.

(1) *Phloridzin poisoning.*—Phloridzin produces glycosuria by its action on the kidney tubules, for if injected into one renal artery the glycosuria occurs sooner and to a more marked degree on that side. Consequently there is no excess of sugar in the blood, but rather a deficiency. For some time no clinical equivalent of this was recognised, but lately cases have been recorded without excess of sugar in the blood which were uninfluenced by diet. The condition has been called renal glycosuria and it has been compared to that produced by phloridzin. A case recently described by Dr. Garrod warns us, however, against coming to this conclusion hastily. A boy was admitted with "renal glycosuria," but his elder sister was in hospital at the same time with typical diabetes. This hardly suggests that it is an entirely separate entity. But at any rate it is established that glycosuria can exist without hyperglycæmia, and I have a case at present under my care with definite hypoglycæmia.

(2) *Excision of the pancreas.*—The connection between pancreatic disease and glycosuria had been established clinically before von Mering and Minkowski showed that in dogs excision of the pancreas is always followed within twenty-four hours by glycosuria, which reaches its maximum on the third day. If a portion of the gland is left it can control the glycosuria even when its connection with the duodenum is severed, but according to Hédon only when it can pour its internal secretion direct into the portal circulation. For then the sugar is on its way to the liver—that great carbohydrate storehouse. Knowlton and Starling found that whereas the heart of a normal dog consumed sugar at a definite rate from the blood, the heart of a depancreatised dog consumed little or no sugar until pancreatic extract had been added. This definitely confirmed the general impression that the internal secretion of the pancreas is necessary to the utilisation of sugar by the tissues. Whether that secretion is the product of the pancreatic cell-islets we need not stop now to consider.

Rose Bradford has recorded a case where a large pancreatic tumour was seen at an exploratory laparotomy; three years later the patient was found to be suffering from severe diabetes, and the tumour could no longer be detected. Recently I had a case illustrating the converse condition. A woman who had been admitted for diabetes returned three years later with all the signs of obstruction of the common bile-duct and the pancreatic duct. As she was sixty years of age I suspected new growth of the head of the pancreas, but under treatment the obstruction disappeared, though the glycosuria persisted. Such cases suggest that diabetes may have a pancreatic origin,

even though there is no evidence of disease of the gland at the time the glycosuria is observed.

Systematic use of the tests for pancreatic insufficiency will in future enable more of such cases to be recognised.

(3) *Adrenal glycosuria.*—Injections of adrenalin are known to excite glycosuria, as when the drug is used for asthma. Von Noorden regards the chromaffin system, of which the adrenals are the most important part, as antagonistic to the pancreas in regard to sugar metabolism. This antagonism has been used by Loewi as a test for pancreatic disease. Normally the instillation of a few drops of adrenalin solution into the conjunctival sac does not dilate the pupil, but if there is pancreatic inadequacy it does so in about twenty minutes. The normal balance between the internal secretions of the pancreas and adrenals is disturbed. This may also prove a help in determining whether a given case of diabetes is of pancreatic origin. I have found that the chief drawback to the test is that it is too sensitive. The pancreas may be only secondarily involved, and yet the test will be positive. Thus in the case of obstructive jaundice I found the test to be positive, and concluded that the obstruction originated in the pancreas. But the post-mortem examination revealed primary new growth in the liver, while there was merely secondary pancreatitis.

Organic disease of the adrenals does not cause glycosuria, because there is deficiency of the internal secretion. Indeed in Addison's disease there is increased tolerance for sugar. But it has often occurred to me that chronic over-action of the adrenals may afford a possible explanation of the frequent association of glycosuria with high tension in men in later middle life. For both these can be produced by excess of adrenalin. The influence of the nervous system in inducing this will be referred to later.

(4) *Thyroid glycosuria.*—Thyroid extract can excite glycosuria. Myxœdema is associated with increased sugar tolerance, while in Graves's disease sugar tolerance is lowered, and there may be actual glycosuria. Hence the myxœdematous patient becomes stout, while the sufferer from exophthalmic goitre emaciates. But even in myxœdema the administration of thyroid extract may cause glycosuria. Garrod found it in four out of eleven cases. The proper dose of thyroid extract in myxœdema is one which does not lower sugar tolerance appreciably below the normal. The drug is not suitable for the treatment of obesity, as it may convert a latent into an active glycosuria, for many stout subjects are on the verge of glycosuria. According to von Noorden the internal secretion of the pancreas is inhibited by the thyroid, hence the glycosuria which follows thyroid excess.

(5) *Pituitary glycosuria.*—The secretion of the posterior lobe of the pituitary body is a potent factor in controlling carbohydrate metabolism. In the active stage of acromegaly there is always lowered sugar tolerance if looked for, and frank glycosuria has been found in more than one third of the cases. But with the progress of the disease the posterior lobe is destroyed, and the sugar tolerance rises, while the blood-pressure falls as less pressor substances are produced. For in the earlier stages Humphry and Dixon have found pressor bodies in the urine. In a case of mine which I have followed for six years acromegaly became arrested, and the sugar tolerance was then found to be very high, for she was able to take 250 grm. of dextrose at one sitting without a trace of it appearing in the urine. The development of this exaggerated sugar tolerance may be delayed by the compensatory enlargement of the thyroid which not infrequently occurs. As Cushing points out, hypo-pituitarism may follow acromegaly just as myxœdema may follow exophthalmic goitre. "Frolich's syndrome," on the other hand, results from a primary hypo-pituitarism. There is excessive deposit of fat, hypoplasia of the genital organs, and loss of the pubic and axillary hair. There may be symptoms of pituitary tumour, such as somnolence, headache and bi-temporal hemianopia. Turney has pointed out the interesting fact that in the female the sexual fat atrophies, though there may be excessive obesity elsewhere. For our present purpose the important point is that there is greatly increased sugar tolerance, which is one factor in inducing the obesity. Cushing and his fellow workers advise the administration of pituitary extracts in Frolich's syndrome, and suggest that the appropriate dose can be determined by seeing what amount is necessary to reduce sugar tolerance to its normal level again. The secretion of the posterior lobe of the pitituary body appears to excite glycosuria in exactly the same way as thyroid extract,

namely, by inhibiting the internal secretion of the pancreas.

(6) *Puncture glycosuria.*—Recent observations have tended to alter considerably the significance attached to Claude Bernard's classical experiments. It was soon found that puncture of the fourth ventricle excited glycosuria by setting up some nervous irritation, which was believed to act directly on the liver. Then it was noted that previous section of the splanchnic nerve or painting nicotine on the ganglia concerned would prevent the puncture from producing its usual effect. And now it appears that the nervous impulses excited pass, not to the liver direct, but to the adrenals, causing them to throw more of their secretion into the circulation. In short, puncture glycosuria is adrenal glycosuria. The clinical equivalents of this process are seen in the glycosuria of concussion, cerebral tumours, cerebral hæmorrhage, pineal cysts and the like. But we must remember now that there may be another way in which these conditions may excite a temporary glycosuria, and that is by pressing on the posterior lobe of the pituitary body, and squeezing an excess of its secretion into the cerebro-spinal fluid.

I have come across several instances of cerebellar hæmorrhage, which on account of glycosuria have been mistaken for diabetic coma. In one case, which was typical of the others, a man walking along the street suddenly felt so faint and giddy that he had to cling to some railings. He was taken to a doctor, who gave him brandy and injected strychnine, whereupon he became unconscious. When he was brought to the hospital comatose, the house-physician passed a catheter and found sugar in the urine. He was treated for diabetic coma by bleeding and infusion of alkalies, but died in a few hours. At the *post-mortem* examination the urine in the bladder did not contain sugar and a hæmorrhage was found in one lobe of the cerebellum, which had pressed on the fourth ventricle. I have seen the same thing in a lenticulo-striate hæmorrhage where the blood had been effused into all the ventricles. In the case here related the sudden onset was unlike that of diabetic coma; but the mistake is very liable to occur in hospital practice when a patient is brought in already comatose, and no clinical history can be obtained. In such circumstances the test for diacetic acid with perchloride of iron becomes of paramount importance, for it will always be found positive in diabetic coma, while it is negative in this type of glycosuria.

(7) *Asphyxial glycosuria.*—In any asphyxial condition glycosuria may occur. This is probably the cause of post-anæsthetic glycosuria, and of the glycosuria of tuberculous meningitis, to which Garrod and Frew have called attention. It appears when the respirations are shallow and grouped. The distribution of the meningitis does not support the idea that the glycosuria results from the pituitary body or the medulla being involved. It is not the deficiency of oxygen but the excess of CO_2 that is responsible. On passing a stream of carbon dioxide through serum sugar is set free. Carbon dioxide can combine with proteins, especially globulins, and in doing so apparently turns sugar out. This supports Pavy's hypothesis that sugar is normally linked to proteins.

A number of drugs may excite a temporary glycosuria, but they do not throw any further light on its pathology. It is worth while, however, to call attention to the special influence of champagne in this respect, because nervous persons are apt to fortify themselves against the ordeal of life insurance examination by its aid, with results that are distinctly unpleasing.

It will be seen that the present conception of glycosuria is that it is usually due to a disturbance in the balance between the internal secretions. In a few cases the defect appears to be at the very door of entry of the sugar into the blood, the intestinal mucosa, but more usually it is further on. The stream of sugar entering the portal vein is carried to the liver, but on the way it encounters the internal secretion of the pancreas, which enables the sugar which is not stored directly as glycogen to be utilised by the tissues. This internal secretion is antagonised by those of the adrenals, thyroid and pituitary, though the first of these appears also to have a direct effect in emptying the glycogen reservoirs. So that the pancreatic defect may be absolute or merely relative, due to overaction of other glands. This explains the frequent failure to find a pancreatic lesion in fatal diabetes. The glycosuria of pregnancy is also probably due to disturbance of this balance, for enlargement of the thyroid is well known to occur in pregnancy, and more recently enlargement of the pituitary has also

been demonstrated. It is noteworthy that the one organ which is most obviously connected with carbohydrate metabolism, the liver, seems to play little part in the pathology of diabetes. As von Noorden says, there are no grounds for assuming that the chemical processes there are qualitatively other than normal.

Treatment.—Having eliminated as far as our present methods will allow any gross disease of organs which may be associated with glycosuria, the ideal to be aimed at is regulation of the diet to a point at which both sugar and diacetic acid are absent from the urine. In a sense these aims are antagonistic, for it is the deprivation or non-utilisation of the carbohydrates that causes the acetonuria. I would prefer the patient to pass sugar rather than diacetic acid, for the latter implies starvation, if not intoxication. No one to-day would think of putting the patient on to a very restricted diet all at once; to do so would be to court the danger of coma; the restriction should be quite gradual, and the urine tested at frequent intervals with ferric chloride for diacetic acid. Even though in a severe case we are unable to render the urine free from sugar, we can probably find a point to which the intake of carbohydrates can be raised without increasing the glycosuria while diminishing the acetonuria. This is the best point at which to maintain the patient's metabolism, for even if further restriction produces a fall in the amount of sugar, it is not beneficial if it causes a return or an increase in the acetonuria. It may be asked why it is necessary to diminish the glycosuria at all if it is merely a symptom of the perverted metabolism. The answer is that damage to the sugar-forming apparatus appears to be partially recovered from under physiological rest, while carbohydrate excess seems to increase the damage, and that the complications, apart from coma, depend upon the hyperglycæmia. I do not intend dealing with the details of treatment; I merely wish to call attention to certain principles which should guide us in the control of (*a*) the glycosuria and (*b*) the acetonuria if present.

(A) *Control of glycosuria.*—Failure to utilise carbohydrates is rarely absolute, and it is our first duty to find some form of starch which can be assimilated. Some years ago von Noorden advocated the use of oatmeal for this purpose, and it is certainly true that many diabetics can take this well. It can be given in the form of porridge, gruel or oatcake. Some patients appreciate this relaxation of the diet. They often tolerate potatoes fairly well. Potato cakes can replace bread to a certain extent, and fruit sugar (levulose) can sometimes be assimilated up to 50 grm. (approximately 15 drm.) if cautiously given in divided doses of not more than 5 drm. at a time. If more is given than can be consumed by the tissues at once it will be stored as glycogen and subsequently converted into dextrose. Its cost, however, is prohibitive, except for wealthy patients or in emergencies, such as threatened coma. I have seen striking benefit from its use.

Wheat starch seems to be the form of starch least tolerable to the diabetic, and the abiding difficulty is to find a satisfactory substitute for bread. As Osler says: "Of the gluten foods, many are very unpalatable, others are frauds." Under the present system of more moderate restriction of the carbohydrates we need not insist as a routine on a pure gluten bread, and the Brusson-Jeune rolls which are a mixture of gluten with some starch are convenient and palatable. But even then I have found it advisable to intercalate some days on which a gluten or protein bread is given.

The second point in the control of the glycosuria is the influence of meat proteins in exciting it. This, which is insisted on by von Noorden, is often overlooked, and the patient is allowed to take meat freely. As a matter of fact he can often take a vegetable diet, which must contain a good deal of carbohydrate, better that he can manage a carbohydrate-free diet with abundant meat. It may be that the carbohydrate fraction of the meat protein is not well borne, but more probably it is due to the stimulating effect of the meat on tissue metabolism. Von Noorden finds that the order of toleration is meat proteins least, then casein, next cooked eggs, and finally vegetable proteins, particularly glidine, best of all. The best scheme appears to be to alternate days of carbohydrate-free diet with days of oatmeal diet, and with days on which little beside eggs and vegetables are taken. Fast days with rest in bed may also be intercalated. I will give one or two examples of such diets, prefacing them with the remark that every diabetic is a law unto himself in the matter, and that careful investigation is necessary to find a scheme that suits each case.

Vegetable, egg and oatmeal scheme.—In the course of an ordinarily restricted diet a week of the following diets is introduced:

(*a*) For two days a diet of lettuce, cabbage, spinach, veal broth, three eggs, butter, two lemons for lemonade, coffee.

(*b*) For three days a diet of 8 oz. of oatmeal as porridge or oatcake, 4 oz. of butter, 5 eggs, 2 lemons and coffee.

(*c*) Two days of (*a*) again.

At the recent International Medical Congress von Noorden gave several other examples, of which I select one:

Three weeks of restricted diet (*i.e.* no carbohydrates), with the addition of 60 grm. (approximately 2 oz.) of bread.

One vegetable egg day (*vide supra*).

One fasting day; only weak tea, lemon squash or whiskey and soda (in suitable cases) are allowed, with rest in bed.

One vegetable egg day.

Four days of restricted diet alone.

The same scheme begins anew.

A marked increase in tolerance often follows. I have chiefly used the former of these schemes and have noted distinct improvement, the urine becoming quite free from sugar in some cases, at any rate for a time.

(B) *Control of acetonuria.*—Recognition of the significance of acetonuria has profoundly modified our treatment of diabetes. Acetone comes from diacetic acid derived from the breaking down of fats, which must always go on during starvation or even deprivation of carbohydrates. It naturally follows that the most effective treatment of acetonuria is to find a form of carbohydrate that can be assimilated, when the fall in the amount both of sugar and acetone in the urine is often striking. Can anything else replace the carbohydrates in metabolism? To a limited extent citric and glutaric acids and alcohol can do so. Citrates can be added to the alkaline mixture, which should always be given when the diet is being restricted or when acidosis is marked. While bearing in mind the dangers of the alcohol habit, it is nevertheless often advisable to give alcohol to the extent of about an ounce of the pure spirit in the day. Malt liquors, sweet wines, champagne and liquors will naturally be avoided. Alkalies help in acidosis; Spriggs has pointed out that when 2 drm. of sodium bicarbonate are given in the day to a normal individual, the urine becomes alkaline and remains so for twenty-four hours. But if excess of acids is being formed this is not enough, and the amount of alkali that can be taken without neutralising the urine is a rough measure of the acidosis. A more accurate measure is the amount of ammonia in the urine, which can be quickly estimated by the formalin method. If the condition improves under treatment the urine will become alkaline, and the amount of alkali may be gradually diminished without a return of the acid reaction. It is not sufficient to give sodium alone as is often done, for other metallic salts are also being drained from the tissues, and if one metallic salt is given out of proportion to the others it increases the excretion of those others. I employ the following mixture, based upon the proportion between the metals normally present in the urine:

℞	Sodii bicarb. . . .	ʒj
	Pot. citrat.	gr. xxx
	Calcii lactat	gr. iij
	Mag. carb.	gr. iij
	Aq. ad	℥j

Three times a day, and increased up to three hourly according to the severity of the case.

It has been urged against this method that it increases the intensity of the diacetic reaction in the urine, at any rate for a time. But this is merely because the excretion of the acetone bodies is being facilitated, not because their production is being increased. They are now excreted as metallic diacetates instead of in combination with ammonia derived from the breakdown of the proteins of the tissues. This alkaline treatment should never be omitted in severe diabetes.

I have nothing to add to the recognised treatment of diabetic coma when once developed, but much may be done in the way of prophylaxis by careful attention to the acidosis. As soon as coma threatens the rectum should be cleared out, and 3 per cent. of $NaHCO_3$ with 4 per cent. of levulose given by drop enema until about 40 grm. of each have been administered, *i.e.* about $1\frac{1}{3}$ litres of fluid.

It may be objected that I have not drawn sufficient distinction between glycosuria and diabetes, but there is no hard and fast line between the mild and the severe types. A case may pass from the mild to the severe form if unsuitably treated, while less often under careful treatment the severe type may become more amenable. I have tried throughout to give the results of my own experience, but it will be sufficiently obvious how much I, in common with all others interested in the subject, owe to the work of von Noorden in Germany and Garrod in this country.

September 29th, 1913.

METASTASIS OF LUNG SECONDARY TO SARCOMA UTERI SIMULATING PNEUMONIA.

By T. MUIRHEAD MARTIN, M.D., C.M.,
Clinical Assistant, Nose, Ear, and Throat Department, Prince of Wales's Hospital, Tottenham, and Queen's Hospital for Children.

THE clinical aspects of metastasis secondary to malignant disease of the viscera are of considerable interest and importance. When the existence of a primary tumour is obvious, or when such a tumour has been more or less recently removed, evidences of disease in other organs must cause anxiety. But this relatively straightforward state of affairs by no means always exists. The primary growth may perhaps be overlooked, or the history of it may be concealed, or its nature—even though the tumour has been removed by operation—may have been doubtful. Still further, the interval between the primary growth and the secondary development may be so considerable that suspicion is at rest; and in some cases the symptoms of the later growth may be so indefinite that the true meaning may be missed.

The subject has been discussed by Sir William Osler* in his lectures on "The Medical Aspects of Carcinoma of the Breast," and by Dr. C. O. Hawthorne.† To quote from the latter—"With evidences of visceral disease of more or less obscure significance, and possibly therefore indicative of a malignant growth, subsequent disturbances, even though remote in time and situation, and wanting in precision, should be regarded with active suspicion." The following case illustrates some of these points:

L. K—, a woman, æt. 48 years, consulted me in September, 1912, for pain in the right side, cough, and expectoration of blood-stained sputum. The physical signs were those of pleurisy and pneumonia (marked dulness, bronchial breathing, and loss of vocal fremitus), with a temperature of 102° F. Under rest in bed and appropriate treatment she gradually improved. The temperature fell to the normal level and the symptoms disappeared, so that after three weeks she thought she had completely recovered. There remained, however, at the right base physical signs which were regarded as due in part to a thickened pleura, and in part to fibrotic changes in the lung substance. During the next two months she lived in the country, and appeared to enjoy good health.

In January, 1913, however, she again complained of pain in the right side, and was much troubled with cough, attended by the expectoration of sputum deeply stained with blood. The temperature was normal, and examination showed absence of tubercle bacilli from the sputum. On the other hand, the physical signs had become much more marked. Practically the whole of the right chest was dull to percussion, and the dulness passed across the middle line in the front of the chest, and the cardiac impulse and percussion were found outside the left nipple line. In the lower part of the chest the respiratory murmur was feeble or absent, but at the higher levels the murmur had a tubular or bronchial quality. To aid the diagnosis an exploring needle was introduced, but only a little blood-stained fluid was obtained.

These facts manifestly had a threatening aspect, and a careful investigation of the history was therefore made. This showed that she was in University College Hospital under the care of Dr. Blacker, who kindly informed me that "In June, 1910, she had a degenerative fibroid polypus removed, and a second growth in December, 1910, the latter being of doubtful character. In April, 1911, another sloughing tumour was removed, and this had the definite structure of a sarcoma." The uterus and appendages were at once removed.

In view of the history and the clinical facts, secondary sarcoma of the lung was diagnosed. The sequel promptly confirmed this conclusion. The symptoms continued in spite of treatment. Her appetite failed, she rapidly lost flesh, and died of exhaustion on April 20th, 1913.

The remarkable point is the appearance of malignant disease of the lung in the guise of an ordinary pleuro-pneumonia, this diagnosis seeming for a time to be confirmed by the marked improvement in the general condition. Had the history been fully known at this stage, it might perhaps have modified the prognosis. Yet it could hardly have afforded ground for a confident diagnosis of malignant disease.

September 29th, 1913.

* 'American Medicine,' April, 1901; 'Brit. Med. Journ.,' January 6th, 1906.

† 'Studies in Clinical Medicine,' p. 151. Bale, Sons and Danielsson, 1912.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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THE CAUSE AND TREATMENT OF TRAUMATIC VENTRAL HERNIA.*

By W. McADAM ECCLES, M.S., F.R.C.S.

The pressure within the abdomen is positive. Make an incision through the abdominal wall, and the omentum and intestines will protrude. Nature has so built up the parietes of the abdomen that, save at the usual hernial regions, protrusions rarely occur, and when they do, they are usually the result of traumatism.

Lesions of the abdominal viscera, although, perhaps, not more uncommon than they were fifty years ago, have come to be dealt with so frequently by operation that traumatic ventral herniæ are all too common. A laparotomy may be a brilliant achievement in that it has removed a fatal condition from the patient, but it may leave him with an annoyance, and possibly a danger, which is almost worse than death. This is an uncontrollable traumatic ventral hernia.

Causation of a Traumatic Ventral Hernia.

The factors which enter into the causation of hernia after laparotomy may be stated to be:

(1) Improper incisions.

(2) Faulty technique in the closure of abdominal wounds.

(3) Sepsis, primary or secondary.

(4) Increase of intra-abdominal pressure after the closure of the wound.

Let us review these causes *seriatim*.

(1) *Improper incisions.*—An incision may be an improper one because it has been made in the wrong place, or it may be an improper one because the method of carrying it through has not been right.

* A clinical lecture delivered at St. Bartholomew's Hospital.

The abdominal wall is a very complex structure, consisting of planes of muscles, of aponeurotic areas, and of lines of fibrous tissue.

The oblique muscles have their fibres so arranged that obviously they mutually reinforce one another, while the transversalis muscle-fibres lying deep to those of the internal oblique again exercise a restraining force against protrusion of viscera. The rectus abdominis is a most useful structure, especially the fibrous tissue of the superficial and deep layers of its sheath. The aponeurosis of the external oblique and the fibrous tissue of the rectus sheath are of great value in the prevention and cure of traumatic ventral hernia.

The middle line of the abdomen—the linea alba—is composed of the union of the fibrous tissue forming the sheath of the two recti. It is broader on the cephalic side of the umbilicus, where the recti muscles tend to diverge somewhat, and narrower between the umbilicus and the symphysis pubis, where the two recti lie very close to one another.

Extending from the tip of the ninth costal cartilage in a curved line downwards to the outer part of the crest of the os pubis is the linea semilunaris, the meeting-place of the ventral aponeurotic edges of the oblique and tranversalis muscles. This line is placed considerably further out than is often imagined, being at the level of the umbilicus often as much as 3½ inches distant from that depression in the adult. It must also be remembered that the nerves passing to supply the rectus muscle run into the rectus sheath obliquely across this line.

From these rough recollections of the anatomy of the abdominal wall it will be readily seen that incisions planned to expose the contained viscera may be made—

(i) Through the middle line, the linea alba.

(ii) Through the rectus sheath, either displacing the whole breadth of the rectus muscle, or separating the vertical fibres after its sheath has been cut.

(iii) Through the linea semilunaris.

(iv) Through the muscular planes of the two obliques and the transversalis.

(v) By a combination of one or more of the foregoing.

Incisions made in the middle line often have the great advantage of more thoroughly exposing the parts to be dealt with than do lateral incisions. But the linea alba, once divided, is probably never again so efficient a barrier against protrusion as it was before its section. However, it cannot be said that an incision in the middle line of the abdomen is an improper incision; its closure, however, may be very faulty.

Incisions through the rectus sheath, with displacement of the enclosed muscular fibres, have much to recommend them, especially in the prophylaxis of traumatic hernia. But there are two serious objections to them, the one that the muscle may be difficult to retract, particularly when it is well developed and the relaxation by the anæsthetic not complete; the other that one or more of the nerves going to the muscle may be damaged if the muscle is displaced towards the middle line.

Splitting the vertical fibres of the rectus itself has little to recommend it and much to condemn it, for not only are the epigastric vessels liable to be troublesome, but both edges of the rectus fibres require to be retracted, with a probability that they may be severely bruised if not actually torn. Further, sutures will not hold satisfactorily in the edges of the separated muscle-fibres.

Incisions through the linea semilunaris are to be avoided, for they are difficult to close accurately, they almost always damage one or two of the nerves going to supply the rectus muscle, thereby tending to cause atrophy and degeneration of these segments of the muscle, and passing entirely through fibrous tissue they may not heal too readily. Such incisions may, therefore, be deemed improper ones, and are only to be made in cases of absolute necessity.

Incisions through the two obliques and the transversalis muscle should always, if possible, partake of the nature of "grid-iron" or "muscle-split" incisions (Figs. 1 and 2). To divide the fibres of these muscles across the line of their direction courts the formation of a traumatic ventral hernia. If, after the fibres have been separated and retracted, it becomes necessary to obtain further room, it is best to continue the incision between the transversalis fibres inwards into the rectus sheath, and then pull the rectus towards the middle line rather than divide the fibres of the transversalis or internal oblique across their line of direction (Fig. 3).

Again, if the original separation and retraction

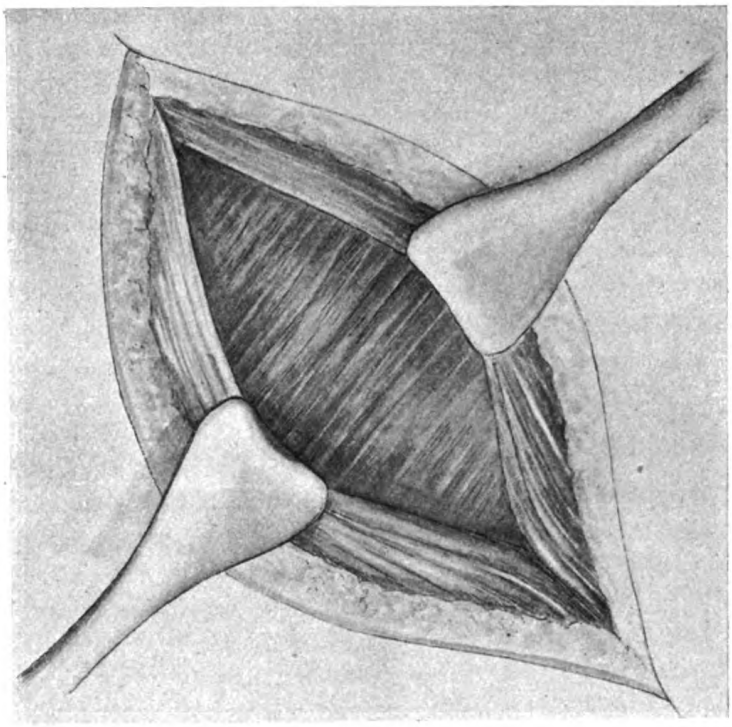

FIG. 1.—To show incision in right iliac region, aponeurosis of external oblique retracted and muscle-fibres of internal oblique exposed.

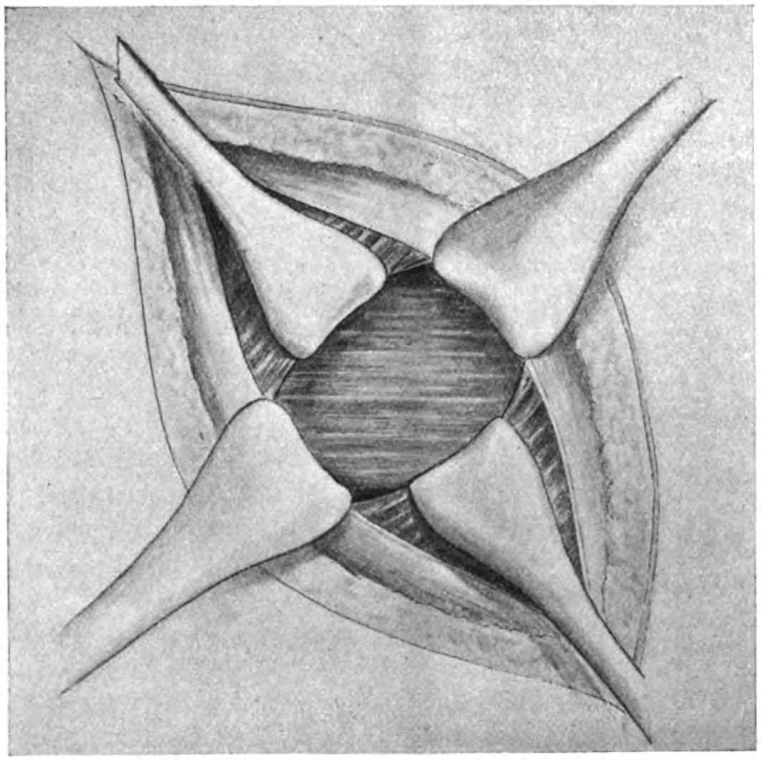

FIG. 2.—To show muscular fibres of internal oblique retracted, and the fibres of transversalis exposed.

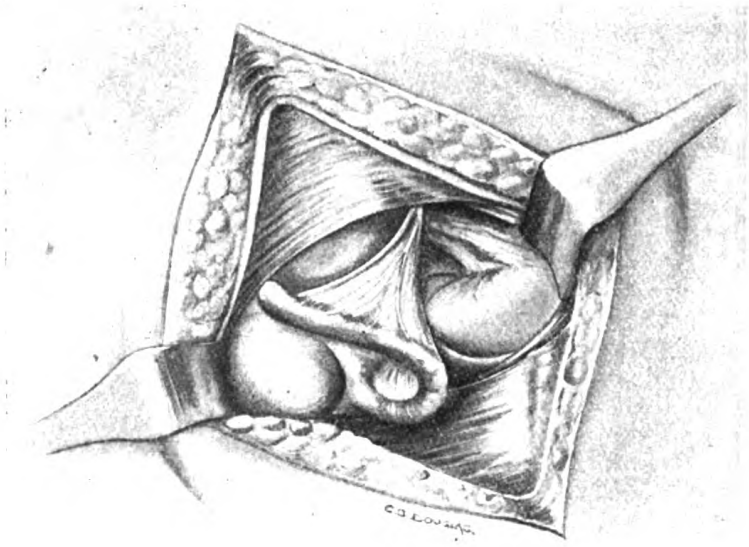

FIG. 3.—To show viscera exposed through "muscle-split" incision in right iliac region.

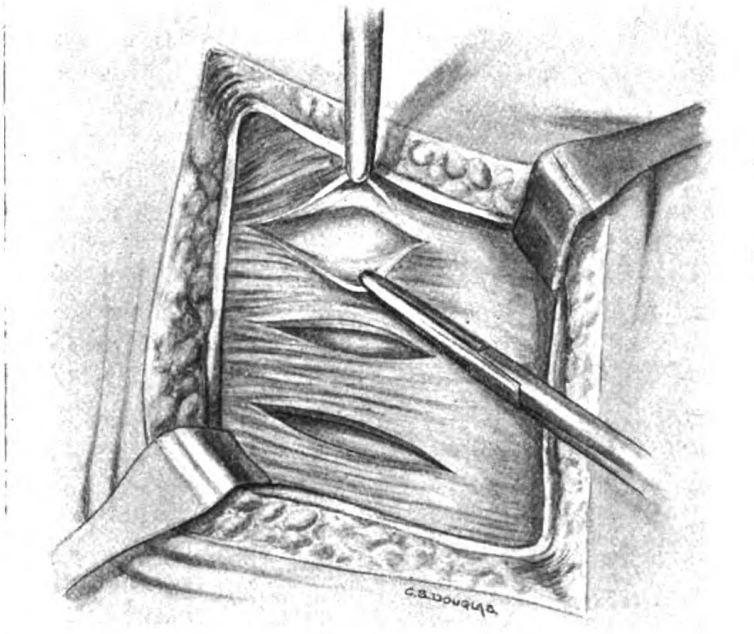

FIG. 4.—To show how several separate separations of internal oblique and transversalis fibres can be used to give more space for deeper dissection.

of internal oblique and transversalis fibres does not give the surgeon sufficient room for, say, a high appendix, or a low tube, then it is much wiser to make another similar but distinct separation and retraction higher up or lower down than to run the greater risk of a ventral hernia by dividing across the fibres of these muscles (Fig. 4).

Incisions involving a combination of any of the above, may be necessary, but they always have an attached risk of a traumatic ventral hernia, and a risk which may be very great. Such an incision is the inverted ⌐-shaped incision for operations upon the bile-ducts. Fortunately, a ventral hernia is not so common in the upper third of the rectus as it is in the lower part of the abdomen, the intra-abdominal pressure being less in the former area.

(2) *Faulty technique in the closure of abdominal wounds.*—While an abdominal incision may be quite properly placed and carried through, its closure may be so faulty as to be followed by a ventral hernia. Take an incision in the middle line: it divides the skin, subcutaneous and fibrous tissues and the peritoneum. When its closure has to be brought about, it is generally easy to bring the peritoneum and superjacent connective tissue accurately into apposition, but the fibrous tissue of the linea alba may retract, and when an attempt is made to bring it together there is undue tension. Further, the edges of the fibrous tissue which have to be co-apted are not very thick, so that healing is not too sure, even if the suturing is good. It is probably best in these incisions in the middle line to open the sheath of both recti, and to deliberately suture the internal edges of the two muscles together, and then to unite the edges of the superficial sheaths, if possible, by superimposing them (Fig. 5).

A very important point in the prevention of a ventral hernia is the co-aptation of peritoneum along the whole length of the abdominal incision. This is often easier if the incision through the serous membrane is made transversely rather than vertically, as, for instance, in the trans-rectal incision for the removal of the appendix.

The material used for buried sutures is a matter of some importance from the prophylaxis of ventral hernia. Personally, I always use fine twist silk, not thicker than No. 1 gauge. Silk is readily rendered aseptic by ordinary boiling, it is easily tied, for its size it is very strong, it remains in the tissues for a very long time, and if aseptic and not too thick is never seen again.

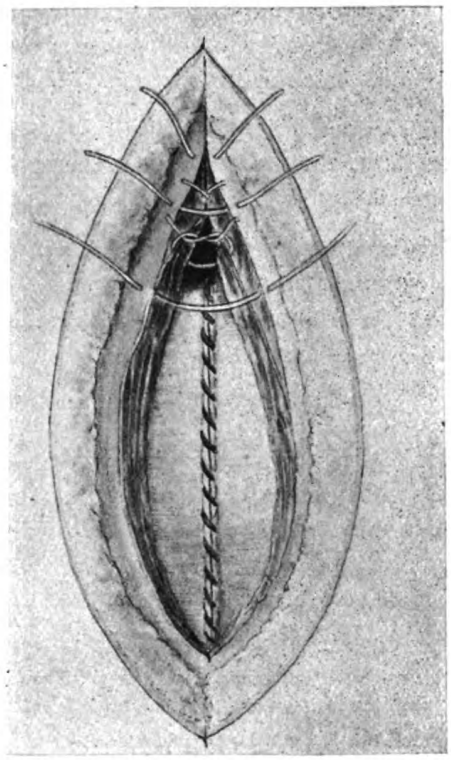

FIG. 5.—To show incision in middle line and method of closure. Deep suture a continuous one in peritoneum and extra-peritoneal tissue. Intermediate one through edges of both recti, and superficial suture through edges of sheath of both recti of the linea alba.

(3) *Sepsis, primary or secondary.*—Sepsis is one of the greatest causes of post-operative hernia. An abdominal wound may require drainage, and often extensive drainage, on account of the primary sepsis for which the operation was performed. Here the occurrence of a subsequent hernia is often unavoidable. If such a protrusion occurs as the result of sepsis in a wound which has been completely closed directly after it has been made, then the surgeon should rightly be blamed for its incidence. There was a fault in the asepsis.

(4) *Increase in the intra-abdominal pressure after the closure of the wound.*—A patient may, on account of pulmonary trouble after the anæsthetic, or on account of accumulation of gas in the intestines, have his intra-abdominal pressure so raised as to cause the edges of the abdominal wound to be under such tension that sutures fail and edges separate.

This is an undoubted cause of ventral hernia, but one which ought to be prevented by due care before and after the laparotomy.

The Treatment of a Traumatic Ventral Hernia.

This is either prophylactic or actual.

Prophylactic treatment.—The facts as related concerning the causation of a traumatic ventral hernia are of themselves sufficiently indicative of the manner in which prophylactic treatment can be carried out.

Here, as in so many other lesions, prevention is greatly to be preferred to cure. There is one other point in the prophylaxis of this complaint, and that is concerning the desirability or not of a patient who has been submitted to laparotomy wearing an abdominal belt or other appliance. There can be little hesitation in saying that where an abdominal wound has been made and closed in a proper manner no external appliance is needed, except under those conditions where the intra-abdominal pressure is likely to be greatly raised.

But there are a considerable number of cases in which some doubt is felt as to whether the abdominal wound has been so closed as to negative any likelihood of a ventral hernia. In such cases the wearing of a well-made and well-fitting abdominal belt, at any rate for the first six months or a year after the operation, may definitely prevent the oncoming of a hernia. In certain other cases in which the weak spot in the abdominal wall is very localised and more readily supported by a truss than by a belt, the application of a suitable truss is indicated.

Actual treatment.—When a ventral hernia has actually appeared after a laparotomy its treatment must be undertaken promptly, for not only may the protrusion be a source of endless worry to the patient, but it may be a source of positive danger, even to life. The discomfort arises from the pain caused by the passage of viscera into the sac, by the interference with proper digestion, and by the difficulty in obtaining proper action of the bowels. The danger arises from the strangulation of the protruded intestine by the margins of the opening, often small and rigid, through which the bowel has slipped, or by its strangulation under a band of adhesion within the abdomen, but close to the hernial aperture. Strangulation necessitates an immediate operation in most cases.

There are two methods of treatment for a traumatic ventral hernia, and these are palliative and operative.

The palliative treatment of this type of hernia is the application of a suitable truss. As a rule an abdominal belt, even if it has the addition of a pad to fit the precise spot where the protrusion comes, is not sufficient in retaining the hernia. Therefore it is that a properly made truss of the right type becomes necessary. Each case requires to be taken on its own merits, and a truss adapted to its particular requirements has to be devised and adjusted. In a not uncommon class of case, namely, where a hernia occurs through the wound made for an appendix abscess, a truss with a properly fashioned interstitial plate pad will be found to be most serviceable.

In many instances the continuous wearing of such an external help will rid the patient of his discomfort, and will place him in comparative safety, but in all cases in which an operation is not contra-indicated, an attempt should be made to cure the hernia.

Operative treatment.—In deciding upon operative measures for a traumatic ventral hernia several points have to be borne in mind. The first is as to whether there is a fair chance of complete cure by operation. In connection with this the size of the opening, the amount of tissue surrounding it and the conditions of the muscles near are important factors. The second is whether the operation is fairly free from real danger to life. In connection with this it must be remembered that the operation involves the opening of the peritoneal cavity, that nearly always adhesions between omentum or intestine and the sac-wall will be encountered, and that there is the possibility that intestine may have to be resected. With due care and experience in operating but little risk should be run by the patient.

Having determined upon operation, the line of the incision has first to be decided. If the contents of the sac are completely reducible, they should not be reduced until the position of the incision has been delineated. Generally it is best to make a curved incision around or near the base of the protrusion, gradually deepening it until the peritoneum is reached. In this way any danger of

inadvertently damaging adherent viscera will be avoided.

As soon as the interior of the sac has been opened, adhesions should be looked for. These may be between the contained viscera and the sac-wall, or between portions of different viscera themselves within the abdomen. The operator's skill may be sorely taxed in dealing with these adhesions; patience, care and time will have to be expended upon them.

Having freed and reduced the contents of the sac, the superfluous peritoneum is cut away, and the edges of the aperture in the peritoneal wall drawn together with a continuous suture of fine silk. In some cases this proceeding is quite easy, in others most difficult, partly on account of the peritoneum being very fragile, and partly on account of tension. It may be that interrupted sutures will accomplish the closure more readily than a continuous stitch.

Then comes what is perhaps the most important part of the whole of the proceeding, namely, the overlapping of the tissues forming the muscular and aponeurotic planes of the abdominal wall. It is essential that these tissues should be super-imposed, and it is not enough that their edges be merely brought into apposition and sutured together. A little dissecting up of the planes renders the overlapping more easy, but it may be necessary in some instances of a rather large gap to slide, as it were, a flap from an adjacent area over the aperture (Figs. 6, 7 and 8). It is always well to remember that, whereas muscular tissue is highly useful as a protective against a recurrence of the protrusion, it does not hold sutures well, and they are apt to cut out, especially when the muscle is put into action. Hence, aponeurotic tissue is of great service, and wherever possible it should be brought in front of a muscular plane. If it is possible to get more than one layer of aponeurosis super-imposed, the success of the operation is almost certain.

The operator is sometimes faced with cases in which it is impossible to get the natural tissues of the abdominal wall even to meet at their edges, still less to lie over one another. In such cases the use of silver filigree may be desirable. The action of this material is two-fold. It serves, as it were, as a scaffolding on which the new connective-tissue

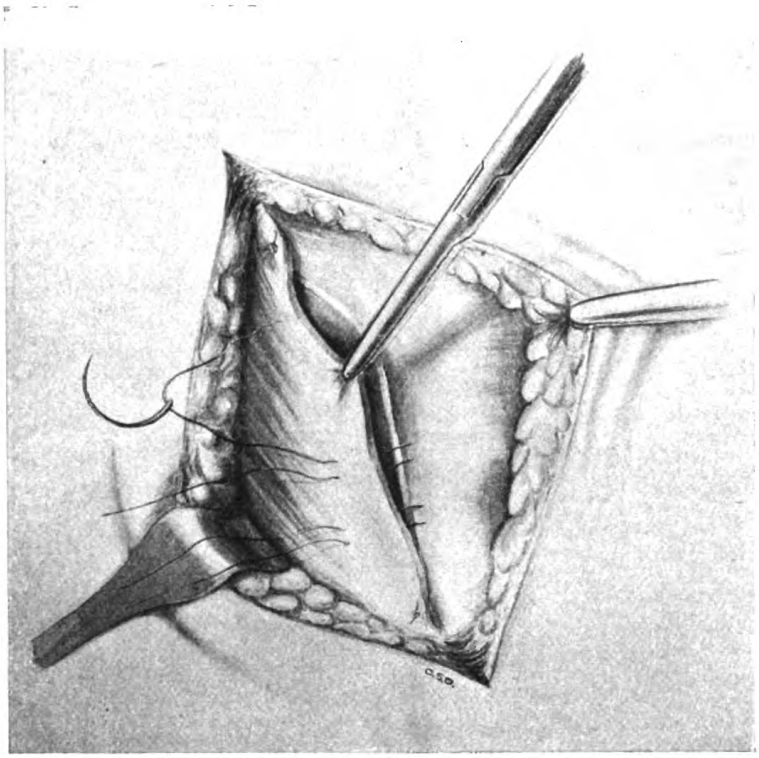

FIG. 6.—To show two layers of muscle and aponeurosis being made to overlap by mattress sutures.

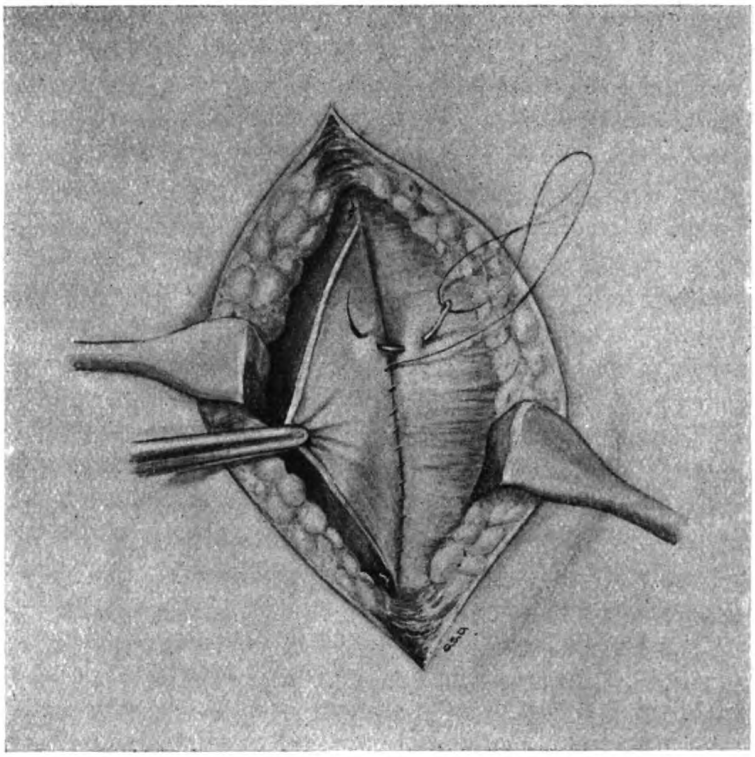

FIG. 7.—To show a continuous suture fixing the deep surface of the superficial flap of aponeurosis.

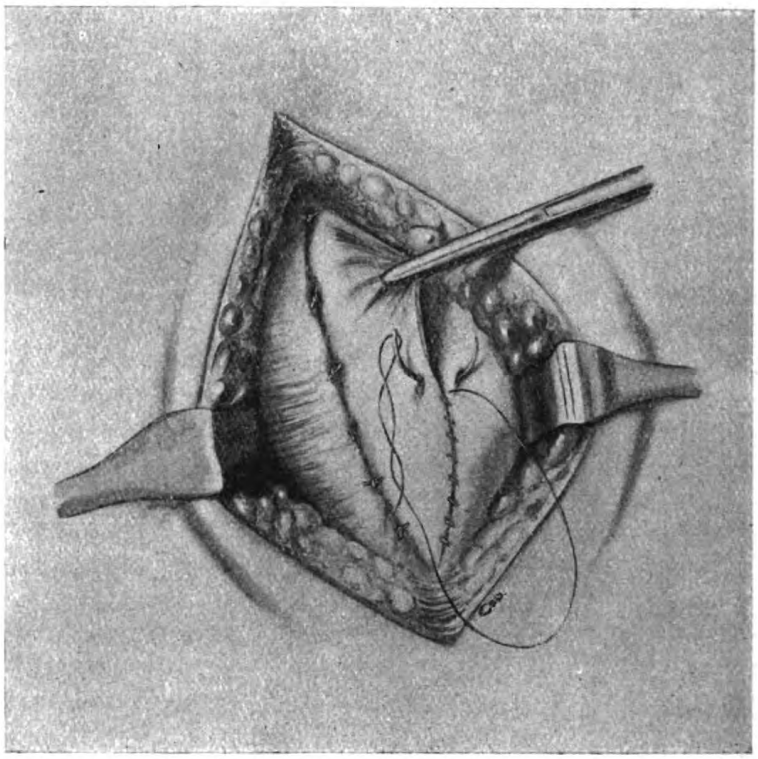

FIG. 8.—To show the final continuous stitch fastening the superficial layer in position.

cells can go to work and form fresh material which fills the gap, and it also acts by its very presence as an effectual barrier against further protrusion. If the filigree is made in the proper manner with fine silver wire, and with two lateral as well as a central backbone, and is introduced carefully without any doubling up and there is complete asepsis, it gives excellent results. It is best to place the filigree on a muscular or aponeurotic plane, but it can be quite safely laid upon extra-peritoneal tissue, and even upon the surface of exposed intestine. A suitable filigree should always be ready in the steriliser in every case of operation upon a large traumatic ventral hernia.

October 6th, 1913.

SKIN DISEASES IN GENERAL PRACTICE. By HALDIN DAVIS, F.R.C.S., M.R.C.P., Physician in charge of the Skin Department, Paddington Green Children's Hospital, London. London: Oxford Medical Publications. Pp. 340. 15*s*. net.

Of Dermatology it may truly be said that the opportunities of acquiring a practical knowledge are the gift of the few. The majority of practitioners amass their information by making a mental picture of the patient, and subsequently referring to an atlas or other collection of illustrated skin diseases. The arrangement of the book is on topographical lines: that is to say, skin lesions attacking a particular area of the body are grouped together. In this way are considered skin diseases of the face, scalp, limbs, hands, feet, groins, etc. In tackling an obscure or unknown form, this considerably narrows the field, and by a policy of differential or exclusive diagnosis the particular entity may easily be ultimately recognised. Eczema and syphilis are separately considered at some length, including the modern treatment for the latter, and there is a most informing chapter on "Some Modern Methods of Treatment, including Ionisation, Radium Emanation, Refrigeration," which no modern practitioner can afford to miss. Excellent prescriptions abound, and there are over sixty illustrations. We predict for this work a hearty welcome.

CARCINOMA OF THE STOMACH.

By ALBERT J. WALTON, M.S., F.R.C.S., M.B., B.Sc.,

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CARCINOMA of the stomach is a disease of such frequency and of so high a mortality that little excuse is required for again directing attention towards it. Its frequency is insufficiently recognised, and this in spite of the fact that 4000 people die of it in England annually, and that in 22 per cent. of all cases of carcinoma in men the growth is situated in the stomach (24).*

Since, however, it is very prone to be mistaken for one of the less severe lesions of the stomach, its frequency as regards other gastric lesions is of even more importance. During the years 1903 to 1912 inclusive there were admitted to the London Hospital 3746 cases of gastric disease other than carcinoma, 1126 of which were considered surgical. During the same period there occurred 825 cases of carcinoma of the stomach, 461 of which were operated upon—that is to say, that over one in six of all cases of gastric disease is carcinoma; whilst nearly one in three of all gastric cases operated upon is also carcinoma. If these figures are borne in mind there will be less tendency to treat early cases medically as gastritis, and the greater tendency to realise that no other condition of the stomach should be diagnosed until carcinoma has been as far as possible eliminated.

ÆTIOLOGY.

The disease is most common between the ages of forty and sixty, but no age is exempt, cases having been recorded even in children under ten. Ness and Teacher (17) report a case in a boy, æt. 14 years and 5 months. Such cases are, however, unusual, and in Sherren's (24) series only one was under the age of twenty. After this age they become more frequent, the disease being relatively frequent between the ages of twenty and thirty.

Men are more frequently affected than women. In the London Hospital series above mentioned 548 cases were males and 277 females. This

* The figures refer to the bibliography at the end of the article.

ratio is in very close agreement with that given by Sherren in his own series of chronic gastric ulcer, the proportion there being 60 to 37—a fact which, as will be shown later, has some bearing upon the origin of carcinoma.

Pathology.

Carcinoma of the stomach may be primary or secondary. In the majority of cases the disease is primary, secondary deposits from other viscera being very unusual. At times carcinoma of the œsophagus may be followed by a deposit in the stomach, the spread having in this case possibly taken place along the submucous lymphatics.

In other cases it may involve the stomach by direct spread from other viscera, such as the colon, gall-bladder, pancreas and œsophagus.

Primary carcinoma is generally of the cuboidal celled type, but occasionally it may be columnar. There is to the naked eye no point of distinction between the two; it is only on microscopic examination that the difference can be seen. The growth occurs in several different pathological forms. Most commonly there is present an ulcer, the edges of which are raised, thickened and irregular, whilst the surface is granular and relatively clean.

In other cases there is considerable reactionary fibrosis, so that a hard solid mass is formed, which, if situated near the pylorus, will tend to constrict the lumen and cause pyloric obstruction.

In other cases, again, the fibrosis may be extreme, and the disease, gradually spreading throughout the whole of the stomach, lead to marked thickening and diminution of the capacity of the organ. Such a condition is known as the "leather-bottle" stomach.

It is but fair, however, to state that many authors do not regard this condition as due to carcinoma. Thomson (25) believes that it is a condition of diffuse fibromatosis unassociated with cancer, while Bird (1) has brought forward a certain amount of evidence to show that in some cases it is due to syphilis. Lyle (6) has considered the condition at length, and has fully investigated the reported cases and the literature upon the subject. He collected in all 129 cases; of these 60 only showed evidence of carcinoma.

Not uncommonly the new growth may form a soft mass projecting into the stomach. In this form there is but little fibrous tissue, and the growth may be relatively vascular. This form is the one most likely to show areas of colloidal or mucoid degeneration, giving rise to the so-called colloid carcinoma.

Most frequently the growth is situated either at the lesser curvature or near the pylorus. In this position it will give rise to symptoms of pure carcinoma unassociated with any mechanical factor such as obstruction. Less frequently it is placed at the pylorus or cardiac extremity. The frequency of its situation at the lesser curvature is a fortunate fact in so far that in this position it is more amenable to surgical treatment.

The most important factor in the pathology of this condition is its relationship to chronic gastric ulcer. It was previously taught that ulcer had but little bearing upon carcinoma, Osler and McCrae (18) finding that only four out of 150 cases of carcinoma had a previous history of gastric ulcer.

Since then, however, a great deal of evidence has been brought forward to show that the relationship is much closer. Thus, Mayo Robson (11) found 57 per cent. and Moynihan (14) 60 per cent. following ulcer. Mansell Moullin (9) obtained a history pointing to previous gastric ulcer in over 60 per cent. of his cases. Wilson and MacCarty (28) give the figures as high as 71 per cent. Mayo (12) in his earlier series found previous ulcer in 54 per cent., but in his later series of 216 cases reported by MacCarty (7), 79 per cent. showed evidence of ulcer.

Perhaps the most important series is that reported by Mumford and Stone (16). They traced sixty patients treated for chronic indigestion who subsequently died; 50 per cent. of them died of carcinoma.

Paterson (20) of late has returned to the older view that gastric ulcer is not a precursor of carcinoma, suggesting that the long-continued symptoms may have been due to the carcinoma, and quotes cases of carcinoma lasting one to four and a half years. In many cases, however, the history is of much longer duration than this. In several of my own cases it dates back twelve years, and it is impossible to believe that a condition of such long standing could have been carcinomatous from the commencement.

Thus it seems to be definitely proved that a large number, roughly two thirds, of all cases of carci-

noma of the stomach follow upon gastric ulcer—a fact of very great importance, for, if the condition of ulcer be diagnosed and given adequate surgical treatment, there is a very strong possibility that the more serious disease of carcinoma may be prevented.

The question of the method of spread, especially as regards the early or late complication of other viscera, is of considerable importance in the question of treatment.

In the stomach tissues a large area may be involved by direct spread, but for some reason at present not understood the duodenum shows a remarkable immunity to involvement in cases of gastric carcinoma as well as to primary growth. Thus, W. J. Mayo (13) found in his series only five cases of carcinoma of the duodenum. Not only does it spread in the layers of the stomach-wall, but permeation of the lymphatics may take place relatively early; the mere presence of enlarged glands, however, does not imply that they are carcinomatous; thus, MacCarty and Blackford (8) found 1404 enlarged lymphatic glands in 200 specimens of carcinoma of the stomach. Of these, only 52 per cent. were carcinomatous. There was, moreover, no relationship between the length of time of the symptoms and the presence of secondaries in the glands.

The method of infection of the lymphatic glands is constant, and the knowledge of their anatomical arrangement forms the basis of surgical treatment. The distribution of these glands and their relationships to the lymphatic areas of the stomach have been fully described by Cuneo and by Jamieson and Dobson (5). The question is, however, of pathological rather than clinical interest and need not be further considered here.

Secondaries in other viscera occur relatively late, and since their presence in nearly all cases prohibits surgical treatment, therefore an attempt should be made to arrive at the diagnosis before they occur.

Symptoms.

The symptoms will vary considerably according to the nature and situation of the disease. Three main groups may be considered according to the position of the growth:

(*a*) Those on the lesser curvature or body of the stomach.

(*b*) Those at the pylorus.

(*c*) Those at the cardiac orifice.

Of these the most important is the first group, for not only does it include the greater number of cases, but the symptoms are those of carcinoma uncomplicated by any mechanical factor. Moynihan (14) first laid stress upon the fact that in this variety the disease might occur with one of three types of symptoms: (1) Cases with sudden onset; (2) cases with many previous attacks of gastric ulcer, the symptoms of the last attack gradually changing into those characteristic of carcinoma; (3) cases with a gradual onset of gastric symptoms which are slow and steady in their course.

In the first of these types the patient may have felt perfectly well until the sudden onset of abdominal pain. In some cases this may only be of a few weeks' or even days' duration, and the pain may be severe and colicky in nature. It will be more or less constant, but is increased by meals. The appetite is gradually lost and vomiting may be present.

The following is an illustrative case:

H. S—, æt. 44 years, was admitted to the London Hospital complaining of abdominal pain. He states that he was perfectly well until seven weeks ago. His illness then commenced with a sudden onset of pain in the upper abdomen: it was cramping in nature and doubled him up. This gradually improved, but never cleared up entirely, so that he now complains of more or less constant aching pain which is made worse by food. He vomits about once or twice a week, but the vomiting does not relieve the pain. There has been no hæmatemesis and no melæna; his appetite has considerably decreased.

On examination there is no marked wasting; the upper abdomen is tender and resistant on both sides; there is no lump to be felt.

At operation there was a large hard mass near the pylorus extending up the lesser curvature on both the anterior and posterior surfaces. One or two glands on the lesser curvature were enlarged. The liver and pancreas were free. A partial gastrectomy followed by posterior gastro-enterostomy was performed. The patient made an uninterrupted recovery, and left hospital after three weeks, and had remained well up to date, four months since the operation.

In some cases the disease may be ushered in

with a severe hæmatemesis, or even with a perforation. In one case the first symptom was acute intestinal obstruction, due to a diffuse carcinomatosis of the peritoneum.

In the second type of case, which, as already mentioned, includes from 50 to 70 per cent. of all cases of carcinoma of the stomach, there is a definite previous history of attacks characteristic of chronic gastric ulcer. In the last attack the symptoms gradually change from those of a gastric ulcer to those of the carcinoma. The pain becomes more constant but less severe, and although made worse by food, has not so definite a relation to food as previously, and there is no period of relief between meals. The appetite alters, so that instead of being merely afraid to take food on account of the pain produced, the patient has a definite objection to food.

The symptoms cease to be intermittent, and in place of lasting only for a week or so they may persist for several weeks or months. As time goes on the other characteristic symptoms are gradually added. The following is such a case:

J. S—, æt. 64 years, was admitted to the London Hospital, and stated that he had suffered with indigestion on and off for twelve years. His symptoms would come on in attacks which lasted from two to three weeks, during which he experienced pain in the epigastrium and left side; the pain would come on about two hours after food and last about twenty minutes. Often during the height of the pain he would vomit, and the vomiting would relieve the pain. During these attacks his appetite had been good, but he had been afraid to eat on account of the pain produced. He had never had any hæmatemesis. During the last eight or nine months the pain had been more or less constant, but was much less severe, consisting rather of a permanent ache and sense of discomfort than of true pain. During this period his appetite had completely gone, and he had a marked distaste for food.

On examination he was much wasted. No abnormal physical signs could be found in the abdomen.

The abdomen was opened, and a large mass found on the posterior wall of the stomach near the lesser curvature. It was adherent to the pancreas; there were several enlarged glands. Owing to the fixation to the pancreas both excision and posterior gastro-enterostomy were impossible; an anterior gastro-enterostomy was, therefore, performed.

In the third type the patient has been perfectly well until the gradual onset of gastric symptoms. These commence as a vague dyspeptic trouble, the patient usually being at or about the age of forty. These symptoms steadily increase and are unrelieved or only partially relieved by medical treatment; the discomfort after food gradually increases and persists for a longer time, so that after a short period there is no free interval between the meals. Appetite gradually goes until there is no desire to take any food. In fact there is an actual distaste for food. The patient steadily loses weight and becomes anæmic.

Mental depression becomes a marked feature; vomiting and hæmatemesis may gradually be added.

The danger of this group is that owing to the gradual onset, medical treatment may be persevered with until the condition is past operation.

The following is a typical case:

A. G—, æt. 36 years, was admitted to the London Hospital with a twelve months' history of gastric trouble, having been perfectly well up to that time. The condition commenced with slight discomfort after food—a feeling, as the patient expressed it, "That his food did not agree with him." Even at this time there was noticed a distinct distaste for food. The pain increased steadily in spite of medical treatment, and three months ago vomiting commenced. The vomiting would relieve but not completely stop the pain, and occasionally there was blood or brown material in the vomit. The patient had been steadily losing weight, having lost three stone in the twelve months.

On examination he was definitely wasted and anæmic, with a pulse of 116. The abdomen was sunken, but there was no lump to be felt.

At operation I found a large adherent mass of carcinoma at the lesser curvature, binding down the posterior surface of the stomach to the pancreas, and running up to the œsophagus. The size and the amount of fixation of the mass made both excision and posterior gastro-enterostomy impossible; an anterior gastro-enterostomy was therefore performed.

The wound healed by first intention, and the patient was discharged from hospital four weeks later considerably relieved of his symptoms.

The symptoms may be considered more in detail as follows:

Discomfort and pain.—These symptoms, as I have pointed out elsewhere (26), are often not the first to appear, and may indeed be almost negligible; they are, however, the conditions which most often give rise to anxiety on the part of the patient and cause him to seek advice. They will vary a good deal with the nature of the case. In those cases which have a sudden onset, they are generally more marked than in the other varieties, and the pain may even be of the nature of colic; but even in this case they tend, after a short interval, to become constant, being increased by meals, but rarely disappearing in the intervals.

If such symptoms arise in a patient at or about the age of forty who has previously been healthy, suspicion of carcinoma should be roused, the important fact being that there has been no previous history of dyspepsia.

In the second type of case—that is to say, those which have followed upon a gastric ulcer—the most characteristic picture of pain and discomfort is seen. The patient will have had many previous attacks of pain typical of gastric ulcer; it will have been severe in nature, situated in the epigastrium, but often radiating; it will have had a definite relation to food, being relieved or entirely disappearing before the next meal. In the last attack the pain will become more constant and will be less severe, although at first it will be increased by food. It will not disappear entirely between meals, so that the patient will come to complain of constant discomfort rather than of severe pain, which he may describe simply as a feeling of heaviness in the epigastrium. It is this alteration in the pain, following upon many attacks, which have definitely pointed to the presence of gastric ulcer, which is so suggestive of the onset of carcinoma; since, however, the typical pain of carcinoma may not be an early symptom, it will be seen that there may be other evidence of carcinoma, whilst the patient still complains of the pain found with gastric ulcer.

In the third type of case, where the onset is slower, pain and discomfort may be quite late in their onset, and it is not an uncommon thing for a patient to seek advice after only a few weeks' history of such symptoms, and to present a well-defined and perhaps inoperable tumour in the epigastrium. In such cases, however, careful investigation will reveal the fact that other symptoms have been present for some months. In others there may have been a very slight but gradually increasing discomfort after food, which, together with other symptoms, may date back for a year or eighteen months, but have only been sufficient to cause the patient to seek advice for a few weeks. Even in this group, as in the others, the pain tends to be constant, and it is the constancy of this discomfort, as pointed out by Hale White (2) and Mansell Moullin (10) among others, which persists for the whole day without relief between meals, occurring in a middle-aged patient, and which is unrelieved by a short but adequate course of medical treatment, which is so suggestive of carcinoma.

Appetite.—Change in the appetite is frequent in all forms of gastric disease. In the case of carcinoma this is an early and not uncommonly the earliest of all symptoms. It takes the form of a very distinct loss of appetite. At first there is simply a loss of the healthy desire for food, this loss being generally most marked towards a meat diet, and most in evidence in those cases showing a gradual onset and unassociated with a previous gastric ulcer. In this respect it seems to bear a close relationship to the alterations in the amount of hydrochloric acid present. Although at first there is merely a loss of appetite, in the later stages, this passes on to a distinct abhorrence not only to food but even to the thought of food. It is thus a very distinct contrast to the changes occurring with chronic gastric ulcer, and affords a useful means if present of recognising carcinomatous changes in a case of chronic gastric ulcer. It must be remembered here, however, that the appetite may in this group remain good even when other evidence makes the presence of carcinoma clear.

Vomiting.—Vomiting is very variable in cases of carcinoma, and is only constantly seen in those cases associated with obstruction. In other cases there is often in the early onset of the disease flatulence and the regurgitation of foul and bitter fluid; later actual vomiting may take place. In such cases it usually occurs when the pain is more severe, but unlike the vomiting of gastric ulcer, it does not relieve the pain. Hale White (3) in his

series found that vomiting was present in 87 per cent. of cases, but he states here that it is most troublesome when there is stenosis of the pylorus. He also appears to include all types of vomiting. In my own experience this symptom is much less marked when there is no obstruction, and even if present is rather more of the nature of slight regurgitation than of true vomiting. In certain cases the vomited material may contain altered blood.

Hæmatemesis and melæna.—Most commonly hæmatemesis is absent during the operable stages. If present it usually takes the form of a loss of small quantities of blood continued over long intervals; this blood, being small in quantity, undergoes changes in the stomach, and thus when vomited is often not recognised by the patient, but shows as the characteristic "coffee-ground" vomit. The presence of this vomit is still often described as one of the characteristic symptoms of carcinoma of the stomach; it is therefore important to remember that in a large proportion of cases there may be neither vomiting nor hæmatemesis. If there has been a well-defined previous history of chronic gastric ulcer the patient may have noticed that in one or more of the attacks blood has been brought up; in these cases it will usually have been larger in quantity and therefore bright red in colour. It is, however, being well recognised now that hæmatemesis is not a common symptom even in chronic gastric ulcer.

In a small percentage of cases carcinoma may be associated with the loss of a large quantity of blood, and mention has already been made of the fact that in a few cases this symptom may even be the first evidence of the presence of carcinoma, perhaps one or two pints of blood being lost. It is in such cases as these that melæna may be present, and it will therefore be seen that blood in sufficient quantities to be visible in its altered state in the stools is a rare occurrence.

If the fæces be examined chemically for the presence of blood—the so-called occult hæmorrhage—it is much more commonly found, and is regarded by many surgeons as a valuable guide to diagnosis.

Anæmia.—The presence of anæmia has often been noted in the early stages of carcinoma, and it is worthy of note that it is often marked in those cases where there has never been any visible loss of blood; in fact this disease is one of the pitfalls when dealing with a supposed case of pernicious anæmia. Rutimeyer (23) found that the hæmoglobin was reduced in 82 per cent. of his 200 cases.

Since this symptom may also be present in cases of chronic ulcer of the stomach or duodenum, it will be seen that its presence is by no means diagnostic. If, however, there has been no visible loss of blood, marked anæmia suggests carcinoma rather than simple ulcer.

Loss of strength, emaciation and cachexia are also commonly found in those cases which have an insidious onset, and it is not uncommon for these patients to seek advice on this account, the other factors of slight discomfort and loss of appetite being only elicited on questioning.

The above symptoms refer only to those cases which may be considered as pure carcinomata—that is to say, those that are unassociated with any mechanical factor. In the less common cases where the growth is placed either at the cardiac end or the pylorus there will be added symptoms of obstruction to the inlet or outlet of the stomach. In the former case there will be difficulty in swallowing food and the condition may closely resemble that of carcinoma of the œsophagus. In the latter case there may be evidence of dilatation and hypertrophy of the stomach. Vomiting will more commonly be present, it will take place at relatively long intervals and will be larger in quantity, perhaps containing particles of food which can be recognised as having been taken two or three days before. The vomited material will have undergone decomposition changes in the stomach, and therefore after it has been ejected may froth and effervesce.

On examination of the abdomen the dilated stomach may be seen, especially if it has been cautiously distended; even cautious distension is, however, a dangerous procedure whenever there is a possibility of carcinoma.

Visible peristalsis may also be seen in the hypertrophied stomach-wall, the waves passing from left to right. It has been so clearly pointed out by Hertz (4) and others that percussion and auscultatory percussion are of no value in determining the presence of dilatation of the stomach that nothing further need be said concerning this method of investigation. Both dilatation and the

presence of active peristalsis can, however, be made clear by means of radiography after a bismuth meal.

Differential Diagnosis.

The first difficulty that will arise in early cases of carcinoma of the stomach is the question of diagnosis from other gastric lesions. It has already been mentioned that cases of gastric ulcer tend in their later stages to become carcinomatous, and how often such changes are shown by a definite alteration in the symptoms. If, however, the important point be grasped that all cases of chronic gastric ulcer should be submitted to operative treatment, the importance of such a diagnosis will not be so great. Operation is necessary for both, but it is in the pre-cancerous or ulcer stage that the operation should be undertaken if possible so that the stage of carcinoma may be prevented.

It is of more importance that carcinoma be diagnosed from simple dyspepsia, for here one condition demands immediate surgical treatment, the other requires only medical attention. If the symptoms are of the definite character described above, carcinoma will be diagnosed. In cases of simple dyspepsia there will not be a marked loss of appetite, and the constant slight pain unrelieved by vomiting will be absent. Pain will be present, but is more likely to be severe, to disappear before the next meal, and to be relieved at its height by vomiting. More important than these factors is the age of onset and the question of the previous health. There are many people who, having had several previous attacks of dyspepsia, will reach the age at which carcinoma is common and still have simple dyspepsia. If, however, a patient at this age who has previously had a normal gastric history becomes the subject of well-defined dyspepsia which does not yield to a short course of adequate medical treatment, and especially if the symptoms are of the nature described above, a suspicion of carcinoma should at once be raised.

At what age, then, should carcinoma of the stomach be suspected? Hale White (2), who lays such stress on the above facts, gives it as forty years. I would prefer to place it at thirty-five, and to say that any patient over this age, but having been previously healthy, who develops a dyspepsia which is not readily cured by medical treatment, is most likely to have carcinoma.

In these cases help may at times be given by a test-meal, for with carcinoma the tendency is for the free hydrochloric acid to be decreased, whilst with chronic ulcer it is slightly increased, and with simple dyspepsias generally unaltered. But, as Panton and Tidy (19) have shown, in the more difficult group of cases where there has been a previous gastric ulcer which has become carcinomatous the free hydrochloric acid may still remain high.

Cases of duodenal ulcer do not as a rule present any great difficulty; with them the pain is usually severe, comes on at a relatively long interval after food, is relieved by the taking of food, and not uncommonly occurs at night. The appetite is good and is often increased. Vomiting is unusual, and, in fact, only occurs if the ulcer has led to contraction of the duodenum. A test-meal will in nearly all cases show a definite increase of free hydrochloric acid.

Gall-stones may not uncommonly give rise to a good deal of difficulty. For many years the symptoms of such calculi may be wholly gastric in nature, but here there will be a feeling of distension and actual flatulence coming on before the meal is finished. Later there will be attacks of more severe pain radiating from the right hypochondrium and epigastrium to the back and shoulder; it is constant during the attacks, but is increased by meals. As I have previously shown (27), this is probably due to the presence of cholecystitis. It is this condition which may be mistaken for carcinoma, but the long previous history of distension after meals with the localisation of the pain, and perhaps the presence of superficial tenderness in the posterior part of the right twelfth segment, will often help to make the diagnosis clear. If the calculi have passed into the cystic or common ducts, giving rise to colic or jaundice, the differentiation of the two conditions will be free from difficulty.

Cases of chronic appendicitis may at times, as Paterson (21, 22) has so clearly shown, give rise to gastric symptoms, but in these cases the patient is usually younger, the symptoms generally date back to childhood, and often appear in definite attacks, lasting one or two weeks. There may also be more acute attacks with vomiting, and the pain definitely referred to the site of the appendix.

Early cases of carcinoma may occasionally have to be differentiated from the crises of an early case of tabes, especially if this condition is unassociated with any other symptom. Here, however, the pain is likely to be more spasmodic in character, and is generally much more severe than it is in cases of carcinoma. The development of other symptoms, such as loss of control of the sphincters, alteration

of the reflexes or ocular changes, will determine the diagnosis.

Treatment.

As in the case of carcinoma elsewhere, the only treatment which at present holds out any hope of success is that of complete surgical removal. In order to entirely eradicate the disease by this means it is essential that operation be undertaken early, and the question of treatment therefore devolves into one as to when operation should be undertaken.

In many cases it is necessary than an operation be performed, even when the exact nature of the disease is not clear, but even in these cases it must be remembered that the condition with which the carcinoma is likely to be confused will be one which will also require surgical treatment, and, therefore, it is only the first stages of the operation which can in any way be described as exploratory in nature. It is in order that only these surgical conditions be operated upon that it is so essential to make a careful investigation. There is always a danger in cases like these, which present difficulty in diagnosis, that the preliminary steps of investigation will be slurred over, and the diagnosis left until the time of the operation. If this be done many cases may be operated upon which would have been adequately dealt with by medical treatment, whereas, if a careful investigation be made, almost certainly any medical condition can be eliminated, and the much greater danger of treating a surgical case by medical means also avoided.

The indications for operative treatment may, therefore, be considered as follows:

(1) Any case presenting the above symptoms sufficiently clearly to make a diagnosis of carcinoma probable, provided only that, without mechanical obstruction, there is definite evidence that the disease is not too far advanced to be relieved.

(2) Any case showing definite evidence of pyloric obstruction. Here, even if the condition is not carcinoma, it can only be relieved by surgical treatment; and even if the disease be carcinoma, but so far advanced as to be no longer removable, much relief may be obtained by short-circuiting the stomach and intestines so as to overcome the mechanical obstruction.

(3) Any case in which the symptoms commence after the age of thirty-five, the patient having been previously healthy and not having been relieved by a few weeks' adequate medical treatment.

(4) Any case where there has been previous attacks suggestive of chronic ulcer, these attacks having been relieved by medical treatment, but in the last attack the symptoms have changed from those typical of chronic gastric ulcer.

(5) Any case where a tumour is present but there is no evidence of metastases.

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THE X-RAY DIAGNOSIS OF ANEURYSM OF THE THORACIC AORTA.*

By MAURICE R. J. HAYES, F.R.C.S.I.,

Radiologist to the Mater Misericordiæ Hospital, Dublin.

IN discussing the diagnosis and physical signs of aneurysm of the thoracic aorta, Professor Osler says: "The clinical picture of aneurysm of the aorta is extremely varied. Many cases present characteristic symptoms and no physical signs, while others have well-marked physical signs and no symptoms. As Broadbent remarks, the aneurysm of *physical signs* springs from the ascending portion of the aorta; the aneurysm of *symptoms* grows from the transverse arch" ('The Principles and Practice of Medicine,' fifth edition, p. 782).

Discussing the prognosis, he says: "The outlook in thoracic aneurysm is always grave. Life may be prolonged for some years, but the patients are in constant jeopardy" (*ibid.*, p. 783). In dealing with this disease, therefore, it is incumbent upon us to avail of every means at our disposal to arrive at an early and accurate diagnosis. Examination of the thoracic vessels by a competent radiologist can rarely leave any doubt as to their actual condition. Considering the grave nature of aortic aneurysm, and how important it is to detect it as early as possible, an explanation of the methods of X-ray examination of the chest is of interest.

The thorax and its contents readily lend themselves to X-ray examination, because the lungs, on account of the air which they contain, are transparent to the rays, while the heart and great thoracic vessels are opaque and cast dense shadows. More information is to be gained from the screen, or fluoroscope, than from the negative. The latter

* Read at the Royal Academy of Medicine in Ireland.

is useful only as a permanent record of the actual conditions at the time of examination, but it gives relatively little information, and no idea as to whether pulsation exists in the vessels. Indeed, there are some aneurysms, as I shall explain later, that can be detected by screen examination only, and, as I shall also show, there are sometimes shadows on the plate so closely simulating aneurysm that without a screen examination they might readily be labelled as such.

In order that any screen examination may be of use it is absolutely necessary that the observer should spend from five to ten minutes in total darkness before making it—this increases the sensibility of the retina—and the room in which he makes the examination should admit no light. Yet I have often seen men who have rarely, if ever, seen an X-ray tube in action, rush from bright sunlight into the X-ray room, expecting to see every detail on the screen, and when they do not see them they go away and say that the X ray in the diagnosis of diseases of the chest is over-rated or valueless. Such an attitude is unscientific and illogical. One cannot recognise abnormalities if he does not know what is normal. It takes a large amount of practice to become conversant with the shadows of a normal chest, and in order to be able to give an opinion of any value one must have a large experience of the characteristics of chests that are not only normal but diseased.

THE NORMAL CHEST is best examined with the patient in the erect position. With the screen on the *front* of the chest one sees in the middle line the shadows of the sternum and vertebral column and the great thoracic vessels; at the sides the shadows of the ribs and their interspaces—the ribs produce a lattice-like arrangement.

On the left side the heart casts a dense shadow, and its pulsations are distinctly visible. The shadow of the heart gradually merges into that of the left diaphragmatic arch during quiet respiration, but on deep inspiration the two shadows can be differentiated.

The diaphragm on the right and left sides can be clearly seen, the right arch, with the dense liver lying beneath it, being well defined. It extends about a quarter inch higher than the left. During quiet respiration the diaphragm regularly moves upwards and downwards like a piston—the length of the stroke being from half inch to three-quarter inch, the average excursion between deep inspiration and full expiration being from two to two and a half inches. It is less in the female and in people of short stature with deep chests. Above, the clavicles and the first ribs can be seen.

The mammary glands in the female, especially when they are pendulous or well-developed, cast dense shadows which may be misleading—on moving the gland of course its shadow moves also.

With the screen on the *back* of the chest the same points are clearly seen, except that the heart and diaphragmatic arches, being further away from the chest-wall behind, their outlines are not so well defined. Neither is the lattice-work arrangement of the ribs seen. With the hands raised above the head, and the elbows directed forwards, the scapulæ are drawn to the sides, and do not impede our view. The lung roots can also be seen, but whether the shadows cast in this region were produced by the bronchi or by the pulmonary vessels was a point about which there was considerable controversy for many years. It is now generally agreed that these shadows are produced by the pulmonary vessels.

The right anterior oblique view.—With the screen on the right side of the front of the chest, and the tube on the left side behind, an oblique view of the thorax can be obtained, and in it we can see in detail the posterior mediastinum. This is the position in which to look for aneurysms of the arch and descending aorta, and no examination of the thoracic vessels is ever complete without viewing the chest in this position.

In the left posterior oblique position, with the screen on the left side of the chest behind, and the tube on the right side of the chest in front, the entire intra-thoracic portion of the descending aorta can be studied. This position is also the best for examination of the thoracic portion of the œsophagus for stricture.

In the left lateral position, with the screen on the left side of the chest and the tube on the right side, the antero-posterior diameter of the heart can be determined. In front of the heart is a clear space called the retro-sternal triangle. It is formed by the sternum in front, the diaphragm below, and the anterior border of the heart posteriorly. Behind the heart is another clear triangular space called the retro-cardiac triangle. It is formed by the vertebral column behind, by the diaphragm below

and by the posterior margin of the heart in front; and here one can plainly see bismuth food pass into the stomach, or an aneurysm of the descending aorta if present. In most healthy adults, more especially those living in cities, some enlarged glands around the root of the lung and in the posterior mediastinum can be seen. Such are the radioscopic appearances of the normal chest.

It is with the screen and not from the plate that the diagnosis of aneurysm must be made, and the chest must be carefully examined from the front and from the back, as well as in the right anterior oblique and the left posterior oblique positions.

In the anterior and posterior examinations, as already explained, the shadows of the great thoracic vessels are in line with those of the sternum and vertebral column. A little to the right of the spine is seen a less dense shadow produced by the superior and inferior venæ cavæ.

That part of the aorta formed by the junction of its transverse and descending portions, called by Walsham and Orton "the left lateral aortic bulge," is not very prominent in young people, and it is always less evident in females than in males. Its size and situation must, however, be carefully noted, as it is a common source of error in the interpretation of negatives. If aneurysms are present they will appear as dark shadows to the right or left of the central dark area. They may or may not be pulsating, and their margins will be defined in proportion to the amount of clot which they contain.

Aneurysms of the aorta can, as a rule, be recognised without much difficulty, but it is not always easy to detect those arising from the innominate, the left common carotid, or the left subclavian arteries, because of the converging upper limits of the thoracic cavity and the smaller amount of transparent lung-tissue here situate.

Aneurysms to the right of the vertebral column are, as a rule, on the ascending aorta, those to the left on the descending aorta. A large aneurysm of the arch of the aorta (Fig. 1) may extend both to the right and to the left of the central opacity formed by the sternum and vertebral column, or it may extend upwards towards the root of the neck. Small aneurysms of the transverse and descending portions of the arch will cast shadows to the left of the vertebral column (Fig. 2). The lateral and oblique positions will help to determine the depth at which an aneurysm is situate. The aneurysm is also nearer to that surface of the chest on which, when viewing it, its limits are most clearly defined.

FIG. 1.—A large aneurysm of the arch. It extends both to the right and to the left of the central opacity.

FIG. 2.—Small aneurysm at junction of transverse and descending portions of arch. The chest is viewed from behind.

To give an accurate diagnosis of aneurysm the left anterior oblique view should never be omitted,

and a detailed description of the shadows as they appear on the screen when viewed in this position must be understood. In this view the shadow cast by the heart and great vessels is not at all unlike that produced by the closed hand with the index finger extended, the fist corresponding to the heart, and the index finger to the ascending transverse and descending portions of the aorta. To the right of this is the patient's left lung, and to the left an elongated clear space, in which lies the œsophagus; next the dark shadow of the vertebral column is seen, and outside it, on the observer's left side, is a clear space corresponding to the patient's right lung.

The shadow corresponding to the index finger has parallel sides and a rounded upper end.

In aneursyms of the arch this upper end assumes a dumb-bell appearance, having a rounded head and a neck. A generally dilated aorta, due to atheroma, may give rise to symptoms simulating aneurysm, and its radiographic appearances in an antero-posterior view will very closely simulate one. The diagnosis of this condition can be accurately made only by examining the chest in the oblique position, when the sides of the index finger shadow will be seen to be parallel, and its upper end will not be bulbous.

From an examination of the chests of a large number of cases, some of whom suffered from angina without any apparent cause for their pain, and others, who were thought to have aneurysm, but in whom it did not exist, and many whom I have examined for stricture of the œsophagus, I must say that general dilatation of the aorta is more common than it is supposed to be. It is the rule to find this condition in most men beyond the age of fifty who have led strenuous lives, although there may be associated with it no evidence of the existence of atheroma in the peripheral vessels. It would be of interest to know if the findings in the *post-mortem* room support this view.

Other shadows occurring within the thorax, which may be mistaken for aneurysm, may be produced by:

(1) New growths.
(2) Consolidation of the lung.
(3) Abscess or empyema.
(4) Enlarged glands.

(1) The shadows of new growths are denser than those of aneurysms, their borders are indistinct and ill-defined, and they are not expansile (Fig. 3). That they may be effected by transmitted pulsation must be borne in mind.

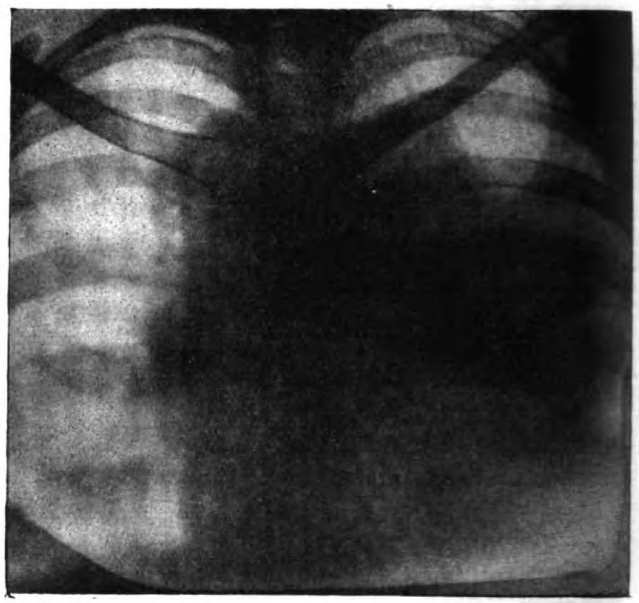

Fig. 3.—Lympho-sarcoma.

(2) A focus of consolidation in the lung, if small and centrally situated, will be entirely surrounded by the transparent lung-tissue, and it will be independent of the great vessels. Expansile pulsation must also be sought for, and the outline of the shadow carefully noted. At the root of the neck it is extremely difficult to differentiate between aneurysms arising from the branches of the arch of the aorta and consolidation of the lung apex (Fig. 4), and in arriving at a diagnosis the antecedent history of the patient, as well as the symptoms and clinical signs present, must be taken into account.

(3) Abscesses or empyema will be recognised by their situation, their shape and the history.

(4) Enlarged glands produce scattered shadows to either side of the vertebral column, and their appearances are so typical that they are not likely to cause any ambiguity.

Another point to be observed in cases of aneurysm is that the long axis of the heart, instead of being oblique, becomes almost horizontal, the base of the heart being depressed and the apex elevated, and this position is independent of any pushing up of the left diaphragm through flatulent distension of the stomach or any such cause.

The position of the heart varies with the position

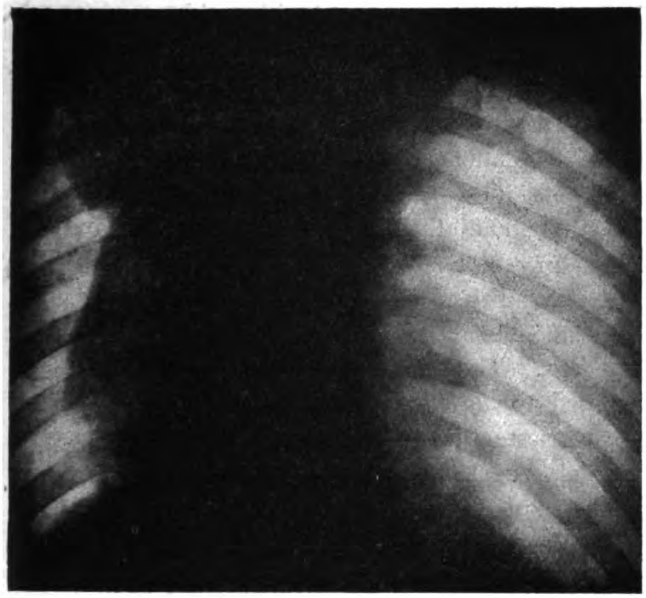

Fig. 4.—Aneurysm of the arch of the aorta with aneurysm of the left common carotid artery.

of the patient, but within narrow limits, and the degree of lateral displacement of this organ on rotating the patient from side to side when in the recumbent position is rarely more than one inch in either direction. One case has been recorded in which the entire heart could be displaced for several inches to the right or left by rotating the patient (Silbergleit, 'Medical Record,' June 20th, 1903). I have seen a freely movable heart in two patients, both of whom were elderly men. In each of these cases, on examining the patient in the erect position, the heart swayed pendulum-like from side to side, and the swinging movements of the heart were synchronous with the ventricular contractions, and they appeared to be determined by them. They were independent of respiration. One of these patients had an aneurysm of the descending aorta just above the diaphragm—a slide of this case I shall show you. The other had a dilated aorta, but no aneurysm. In each case, however, the heart was pulsating rapidly but regularly. This swinging movement of the heart was not seen on examining these patients in the recumbent position. It is a phenomenon which I have not seen described, and I can suggest no other explanation of it than that it was possibly due to an elongated and abnormally free ascending aorta.

October 13th, 1913.

THE SIGNIFICANCE OF CHRONIC COUGH AND WASTING IN CHILDHOOD.*

By E. BELLINGHAM SMITH, M.D., M.R.C.P.,
Physician to Out-patients, Great Northern Central Hospital, and the Queen's Hospital for Children, etc.

It may appear that the title which I have chosen for this afternoon is rather wide and comprehensive. I intend, however, to limit myself to the discussion of cough and chronic wasting in children between the ages of three and thirteen. Again, if you will recall the cases which you have seen in which these symptoms have been the predominant features, I think some of you will agree with me that while these twin symptoms are outstanding features in a number of diseases, they more often at this age are indicative of a common disorder, namely, chronic dyspepsia, chronic intestinal dyspepsia, carbohydrate dyspepsia or mucous disease as it has variously been called.

My excuse for presenting so common a complaint before you is this: it forms at least 50 per cent. of my practice, and I am sure, therefore, that it must form a still greater percentage of yours. It occurs in all classes from the richest to the poorest, while the protean nature of its symptoms is constantly leading to confusion and errors of diagnosis. Let me illustrate this point. In the last few weeks I find that this disease has appeared in my out-patients under the guise of tuberculosis, general debility, consumptive bowels, *petit mal*, heart disease, disorder of the circulation, anæmia and so on. When describing the symptoms I will attempt to show you how these various misappellations have arisen.

First take the cough. It is generally described by the parents as dry, hacking, hollow or irritable cough. Its chief incidence is usually when the child first lies down at night, or when he or she first wakes in the morning. It is unaccompanied by signs in the lungs, is never followed by expectoration whatever the age of the child. This cough, often called rightly a stomach cough, is, I think, not infrequently produced in the throat. If

* A post-graduate lecture delivered at the Queen's Hospital for Children.

you examine the throats of these children, you will usually find one of three conditions: (*a*) Either a shiny condition of the posterior pharyngeal wall, which is in addition covered with a thin layer of mucus; or (*b*) adenoids and slight general enlargement of the tonsils and lymphoid tissues of the pharynx generally; or (*c*), in the worst cases, chronic swelling and injection of all the glandular tissues of the throat, with which is associated a quantity of thick muco-purulent secretion which is constantly trickling down into the stomach.

Secondly, we have the wasting; this may be of any degree, from a slight loss of weight, only appreciated by an anxious parent, to the most extreme emaciation. In the latter case the skin, which is often dry and hairy, is stretched tightly over the bony framework, while the abdomen is not infrequently moderately distended, and the coils of intestine can usually be readily felt or even seen through the thin abdominal wall.

The third distinctive symptom, and one which sometimes has to be elicited by question, is pain. This is usually referred to the epigastrium, but also may be felt in the left hypochondrium, or occasionally over the hepatic region. Sometimes the pain is colicky in nature and is then felt vaguely all over the abdomen.

The next features which point to a derangement of the digestive functions are sundry disorders of appetite and of the bowels. Thus these children, while they may have an ordinary appetite—and I think this is unusual if careful inquiry is made—more often have no appetite at all, "pick over their food," as the poorer mothers say, or have a very large, almost insatiable desire for food. Occasionally the appetite is perverted, there exists only a longing for sour, or highly seasoned foods, or for one particular type of diet such as carbohydrates.

The condition of the bowels is also variable; there is as a rule constipation, more rarely there is alternately diarrhœa and constipation; sometimes the bowels act immediately after meals or even during the ingestion of food. Often the stools contain a quantity of mucus or "jelly and slime" as the mothers phrase it. When this condition is marked you will not infrequently find that thread-worms are also present.

These children complain of nausea, but sickness as a rule is rare. The breath is at times offensive, the teeth not uncommonly carious, while the tongue is large, flabby, and covered with a thin greyish or brownish fur.

Having dealt with the cough, the wasting and pain, and the other symptoms more definitely related to digestive derangement, we come to a variety of symptoms, all of which are not present in every case, and some of which are at times more prominent than others.

The majority of these symptoms owe their origin to an enfeebled nutrition of the various systems of the body. Thus in cases where the child is predisposed to functional nervous disorders, or the nervous system has been directly starved, we have complaints of sleeplessness, teeth-grinding, somnambulism, enuresis, night-terrors, etc. These may be predominant features in any given case so that the child is soaked with bromides or belladonna, while the real *fons et origo mali* is neglected.

Involvement of the vaso-motor system is revealed in fainting attacks, whence the diagnosis of epilepsy and heart disease, in attacks of pallor, "going blue round the mouth," cold hands and feet, purplish swellings under the eyes, and lastly profuse sweating, which is generally most marked at night. This latter in conjunction with cough and wasting suggests to many minds an undoubted diagnosis of tuberculosis, despite the fact that there are no physical signs indicating the presence of that disease. It is well to remember that sweating may occur in any child whose health is enfeebled.

The muscular system of these children is ill-developed and flabby, the disability in this direction being shown in an inability to undertake any exertion, a constant feeling of tiredness, a desire "to lie about." The atony of the bowel is indicated by the constipation, the coils of intestine distended by flatus, and a rarer condition to which not much attention has been drawn, namely, dilatation of the stomach. The skin of these children is generally sallow and rough and dry to the feel. The apparent pallor frequently leads to a diagnosis of anæmia and exhibition of iron, which increases the dyspepsia. In the same way the cough and wasting invariably calls forth a jar of cod-liver oil and malt, which in nine cases out of ten by inhibiting the gastric digestion still further intensifies this distressing condition. The remedies which I think you will find most efficacious in this complaint are preparations on the following lines, viz. a mixture containing tinct. nucis vom. ♏ij–iv, sod.

bicarb. gr. v–x, tinct. rhei co. ♏iv–viij, made up with a little glycerine, and if the child does not object some vegetable bitter such as inf. gent. drm. ij–iv. This prescription you would give about fifteen minutes before meals, and the meal itself is then best followed by some malt extract, drm. j–ij. Where there is lienteric diarrhœa a minim of the liquor arsenicalis alone or in combination with a minim of tinct. opii given just before meals is generally all that is needful. The various nervous symptoms can be met by the addition of a small dose of bromides or of belladonna to the original prescription.

In every case of this nature, the teeth, whether first or second dentition, should be most carefully attended to. Neglect of the first teeth is in a great many cases the starting-point of this disorder. Where possible all early caries should be eradicated and the teeth filled, and where there is oral sepsis and much decay the teeth should be extracted and some simple mouth-wash applied. The unhealthy condition of the throat can be speedily cured by the application of equal parts of glycerine of tannic acid and glycerine of boric acid, breathing exercises, and education in the use of a pocket handkerchief. When threadworms are present the administration of santonin grs. j–ij given on an empty stomach before breakfast and followed three to four hours later by a dose of castor oil is generally sufficient. If necessary this dose may be repeated every fourth or fifth day until worms cease to appear.

But in prescribing these drugs you must remember that the all-important point in treatment is the regulation of the hours of feeding and the nature of the food given. Meals must be at absolutely regular hours, and no food, sweetmeats or raw fruit should be partaken of in between times. All spices, seasoned foods, cakes, biscuits, sweets, pickles and quantities of raw fruits must be prohibited; jam, pastry, bread (preferably stale) and potatoes must only be allowed in moderation. Fruit may be given after meals, but is better cooked. The child may have as much meat, fish, eggs, milk and vegetables as he cares for. Don't thrust milk puddings and porridge on every child—they don't always agree; but at the same time a degree of firmness must be exercised in compelling a child to eat what at first he may show some repugnance for. The great mass of cases are, in the upper classes, due to unsuitable food; in the lower classes to a want of proper food. In both cases an excess of carbohydrates is generally the determining factor. In some there would appear to be an inability to digest even small quantities of starch.

I am now going to show you three cases which illustrate most of the principal features of this disease. Here is a little girl, æt. 12 years, who looks about eight years old. She is referred from the schools as general debility. She is obviously extremely wasted, and she has a slight cough which is worse when she lies down at night. Notice her sallow, dry, hairy skin and wizened features; her general appearance might well lead one to suspect tuberculosis. If you examine her, however, I think you will agree with me that there are no signs indicative of that disorder. I think we can presume on the evidence we have that there is some grave defect either in the diet she is receiving or in her method of digesting and assimilating it, and that she is in fact an extreme case of chronic intestinal dyspepsia.

Here is another little girl, who is wasting, has a bad cough, worse at night and on first rising in the morning, her appetite is very bad, she has pains across the stomach, and has recently suffered from threadworms. She is very thin and poorly developed, but again there are no abnormal physical signs indicating any disease of the various systems of the body. She has been under treatment for two months, but is, as you see, still far from well. You will find, especially amongst the lower classes, that progress in this disease is not infrequently slow. A condition as bad as this child has arrived at probably takes many months or even years for its consummation; you therefore cannot expect to cure it in three weeks.

This boy, æt. 8 years, is still another example of this complaint. He is brought to hospital because he is wasting, has a cough and no appetite, sweats a lot at night, has pains in the chest, is always tired, and has no energy and wants to lie about. His bowels are very constipated, and when they act the stools are full of jelly and slime. There are no worms, but he talks in his sleep, grinds his teeth at night, and has recently shown a tendency to walking in his sleep. When you inquire into his diet you find that he won't eat meat, lives chiefly on bread, likes savoury articles like fried fish and potatoes, and spends a good many farthings on cheap sweets.

As in the previous cases there is absolutely no evidence of any organic disease to account for the wasting and other subjective symptoms, and I should add that in all these three cases there is no X-ray change evident in the thoracic viscera.

You may say, Are not these children perhaps cases of latent tuberculosis of which one hears so much nowadays, and if so have you used the more modern methods of detecting tubercle? I have told you that we have examined the chests in all three cases with the X rays and have found nothing abnormal, and we are then left with the question of diagnosis by means of tuberculin. The two methods at present in vogue are von Pirquet's cutaneous reaction and the injection of old tuberculin. The first I think you will find so unreliable after the age of two or three years as to be of little or no value. The second method, if you give it in the way generally recommended, namely, in three increasing doses given at intervals of forty-eight hours, will prove a number of cases to be tuberculous which never have and never will show any sign of active disease. My advice to you is to regard these methods as mere accessories in diagnosis, and not to pin too great faith to them. If you follow out the lines of treatment I have advised above, you are less likely to make a mistake than you are if you should submit these children to a course of tuberculin merely on the evidence of a positive von Pirquet or tuberculin reaction. The latter course of treatment I have seen utilised in more than one case of this nature, with what could hardly be called favourable results.

Now you will want to know what conditions you can confuse with this disorder—in other words, what other diseases at this age may give rise to a complaint of cough and wasting. One would expect that where these two symptoms were very prominent, it would be natural to mistake these cases for some disorder of the lung. I have told you that the common error is to class these children as tuberculous, they have been called the pre-tubercular—I think most of us are that; while some enthusiasts have made them out to be the subject of tuberculous bronchial glands.

Suppose we consider briefly the tuberculous affections of the lungs in children. They can be classified under four headings:

(1) A general miliary tuberculosis.

(2) An acute confluent tuberculous broncho-pneumonia.

(3) A chronic apical type of adults (very rare).

(4) A tuberculous enlargement of the bronchial and mediastinal glands, with later a secondary invasion of the lung by direct extension.

The first two groups do not concern us here; the symptoms are not chronic cough and wasting, but very acute general infection with death in a few weeks. The third type is so rare that I think I can safely say that although I have examined many thousands of sick children I can still count the number of cases of this nature on my fingers. I had hoped to show you a small boy who has been under my care this past two years with this type of pulmonary tuberculosis, but he has recently been sent to Bexhill. He has all the typical symptoms, viz. a failure to gain in weight, but on the other hand there is not the marked wasting that you have seen in these other children; there is cough and expectoration, and, very important, tubercle bacilli in the sputum. He has bouts of temperature and increased pulse-rate, while the physical signs are those usually present in adults, namely, flattening of chest below right clavicle, impairment of movement, dulness on percussion, high-pitched breathing, and a few moist *râles* over the same area. The disease seems in his case to be chronic; there has been only a very little progress in the two years that I have known him. This, in my experience, has been the trend of most of the cases of phthisis of the adult type in children.

These symptoms and facts are well borne out by this little girl which my colleague Dr. Owen kindly sends us. You will notice that she has been the subject of a cervical caries, and has also signs at the right apex of a very similar nature to what I have told you above, only there are no moist sounds and the child seems very well. She has apparently had no medical treatment for a year, so that for the present we must presume that the disease is arrested. With regard to the fourth group, viz. tuberculous bronchial glands, these also may give rise to symptoms of persistent cough and wasting. In addition they give rise not infrequently to other signs which may assist you to arrive at a correct diagnosis. These are:

(1) A cough which is paroxysmal and rather like whooping-cough without the whoop or vomiting.

(2) Dulness behind the manubrium sterni ex-

tending to the right or left of that bone, and an impaired note between the scapulæ behind.

(3) Distension of the veins of the neck and upper part of the chest and shoulder region.

(4) A venous hum at the junction of the clavicle and manubrium, increased by extending and decreased by flexing the head.

(5) Occasionally a pulmonary systolic murmur.

(6) Pressure on a bronchus, usually the right side, causing an impaired entry over a portion of the lung, in long-continued cases giving rise to signs simulating phthisis.

(7) In more advanced cases there may be pressure on the lower end of the trachea, associated with a stridor which is increased by extending the head and decreased by flexing it.

(8) Where nerves are involved there may be some hoarseness of the voice.

There are other signs, but these are the chief. You must remember that not all these symptoms are necessarily present in every case, and also that singly these signs do not indicate the presence of enlarged mediastinal glands. Where cough and wasting exist, however, with two or more of these signs, it is well to remember the possibility of tuberculous bronchial glands being present.

I have two little boys here who illustrate some of these features. This first boy, æt. 8 years, is brought to hospital for persistent cough and wasting. If you examine him I think you will find some impairment of the percussion note between the spine and the right scapula. You will also notice that the veins on the right side of the chest and the external jugular on the right side are prominent and distended. X rays show a considerable area of shadow in the mediastinum and on either side of the pericardium.

This second boy is a very much more marked example. Notice the large swollen veins over the shoulder and chest and the very prominent external jugulars. You should remember that thin, clear-skinned children have plainly visible veins over the chest, but their distribution is symmetrical and never so prominent as in these two cases. This boy has an impaired percussion note and slightly impaired entry over the upper part of the right lung, and in addition there is a distinct venous hum present. He attends hospital for a persistent cough and wasting. Supporting the view that he is suffering from tuberculous bronchial glands, we have: (1) The fact that he has previously had enlarged glands in the neck. (2) The knowledge that his mother has advanced phthisis. (3) The boy has periods of evening pyrexia, and for some months he has failed to make any progress in weight on the most careful dieting, rest and convalescent treatment. Here again the X rays offer confirmatory evidence of a distinct shadow area corresponding to the physical signs.

The next condition which I am going to show you, and which is well illustrated by this boy, æt. 12 years, is one which is not uncommonly labelled pulmonary tuberculosis or fibroid phthisis without any evidence that it is tuberculous. This condition, which is more adequately described under the term fibroid lung, and where there is dilatation of the bronchi or cavitation, fibroid lung, and bronchiectasis, can be shown in nearly all cases to be a sequence of some previous acute inflammation of the lung substance, such as broncho-pneumonia, which has occurred at an early age, either alone or in association with some infective disorder such as measles or whooping-cough. The physical signs in these cases vary with the degree of fibrosis present. When this change is slight, you will find only evidence of a persistent unilateral bronchitis; when it is marked, you will not uncommonly find a considerable degree of deformity of the chest and a displacement of the heart to the affected side. In these latter cases there will be flattening and retraction of the chest-wall, marked impairment of the percussion note, with bubbling *râles* and tubular or amphoric breathing. This boy in question is brought to the hospital because of his persistent cough and wasting; in addition he sweats considerably at nights, and expectorates a large quantity of sputum in the early morning. If you examine him, you will find that both lungs are partially affected with this fibroid condition. In this case repeated examination has failed to show the presence of any tubercle bacilli, and he has had the lung trouble since he was four years old—that is, for eight years.

There are other more uncommon disorders affecting the lungs which may result in chronic cough and wasting. Here is a boy who has been under my care during the past four years. His first trouble was a very severe double pneumonia, which cleared up, leaving a little residual fibrosis. Unfortunately he became the subject of asthma, and

he now for a child has a very advanced degree of emphysema. His principal complaint is cough, wasting and shortness of breath.

In whooping-cough, in addition to the cough there is not infrequently a considerable degree of wasting. No mistake can be made when the typical paroxysm and whoop is heard. When these are not in evidence the puffy face and suffused conjunctivæ with a history of sickness following the cough generally reveals the diagnosis.

There is still one other intra-thoracic cause of cough and wasting which I have to show you and that is heart disease. This tall boy, æt. 11 years, was brought to see me because of these symptoms. His past history revealed no illness of any importance, but he has been noticed to be a little short of breath for some time. If you examine him you find that he is suffering from a moderately severe heart lesion in the form of mitral stenosis. When he first attended hospital there was some considerable dilatation of the heart to the right and there were a number of moist sounds at both bases. The cause of his cough, etc., is at once evident, the cough being due to the temporary congestion of the lungs, and his wasting to the fact that the action of his failing heart was inadequate to the needs of his rapidly growing body. Occasionally you will find a dual cause for this complaint, as in this boy, who you notice is very wasted and has a persistent cough. In this instance the cough is due to a very large pair of tonsils, while the wasting is dependent on a tuberculous peritonitis.

Gentlemen, the moral which I wish you to draw from this lecture is this, that while there are sundry grave disorders in childhood which are the cause of a persistent cough and wasting, in at least 90 per cent. you will find that an intestinal derangement is alone responsible.

October 13th, 1913.

Uterus Bicornis causing Chronic Transverse Position of the Fœtus.—Von Klein reports six cases of uterus bicornis with transverse position of the fœtus treated by version, and one by Cæsarean section. It seems that this uterine abnormality is a frequent cause for transverse position.—*American Journ. of Med. Sciences*, vol. cxlvi, p. 303.

A DEMONSTRATION OF SURGICAL CASES.*

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Injury to the Elbow treated by Operation.

The first patient I wish to show you this afternoon is a young man, æt. 21 years, who, thirteen weeks before he came into my hands, fell, striking his elbow and injuring it. Now, if we want to improve the results which we attain in surgery, the cases which we should tackle are those which we know in the ordinary way yield bad results. And I suppose that fractures about the elbow give worse results than almost any other kind of fracture. So one set to work to try and improve these. We took this young man into St. Thomas's Hospital, and had a skiagram taken of his elbow. This shows that he fractured his olecranon, and dislocated his forearm bones forward. In consequence of the olecranon being fractured, whenever the people at the hospital reduced the dislocation of his forearm bones, these bones would not stay in position, but slipped out again. Therefore six weeks ago I operated upon him, making a long incision over the back of the affected region, so as to see what could be done to improve his arm. Most of you will be aware that some of our poorest results in surgery follow excisions of the elbow, the arms after excision of the elbow being usually deficient in both power and movement. So one set oneself the task of not only trying to improve the results of fractures in the neighbourhood of the elbow-joint, but also to improve the results following upon excision of the elbow. One can improve the results of excision of that joint by retaining the olecranon, for by retaining the olecranon one retains the power in the triceps and in the arm, and so one overcomes the greatest drawback to the operation of excision of the elbow. In this man's case the olecranon had been broken sixteen weeks before, and was drawn up on the back of the humerus, more so than appears in the skiagram. In his case a long incision was made over the back of the

* At the Medical Graduates' College and Polyclinic.

arm and the elbow-joint opened, and in order to get the bone into position properly, the articular surfaces were removed on the lower end of the humerus. Then the olecranon was freed, and secured on to the ulna by a wire. That was the least satisfactory part of the operation. You see in this patient the whole wound has healed by first intention. In wiring the olecranon to the ulna one found that the olecranon was soft and almost of cheesy consistence, so the wire cut through. The olecranon, which was fastened on to the ulna here, is no longer there; it has been retracted somewhat up the arm. That you will be able to see fairly clearly in this skiagram.

Under the skin in this patient one can feel the wire. As it has performed its function, I shall take the wire out to-morrow. The case enables me to show you the use which has been served by retaining the wire. The patient has now got very fair movement of his arm, and it will materially improve still further. He can move it through an arc of 90°, and as he does that a definite grating can be felt in the humerus.

The next patient is a boy, who had a somewhat similar injury to the other patient, the elbow-joint being again the part affected, and here also we tried to improve results by an operation. Fourteen weeks before he came into my care a lorry had passed over his arm and leg. The leg was very much torn, and it was considered at that time that the leg injury was very much the graver of the two. But when the leg got well, it was found that there was very little movement in the elbow, and that his arm was of but little use to him. Therefore we made the attempt to improve that arm. As shown in this skiagram of the elbow-joint, which includes the lower end of the humerus, the radius and the ulna, the injury was a spiral fracture of the lower end of the humerus, with the lower fragment displaced forwards, leaving it in such a condition that when he tried to extend his arm, the ulna came up against the broken bone at the back, so that he could only extend his arm quite a little. The operation was conducted on practically similar lines to the other; a long incision at the back of the arm, the olecranon was preserved, and the movements in the joint restored by removing a portion of the lower end of the humerus only. As I mentioned in reference to the other patient, one hoped in that way to preserve the strength and greater use in the arm than by the ordinarily accepted method of excision, in which latter procedure not only is bone taken away from the lower end of the humerus, but from the upper end of the radius, and the olecranon is removed as well. This boy was operated upon a fortnight ago, and it was necessary to do more to him than to the previous patient. Hence it will take this boy longer to get back his movements than the other patient. But the condition in which you see the arm now will show you this point. Before the operation, he could straighten his arm only a little, because the olecranon came up against the broken humerus at the back. But now he can get his arm straight out, and after it has been well massaged it can be brought up to the shoulder. So that although the operation has been done only a fortnight, he has a range of passive movement from his shoulder on the opposite side to a perfectly straight arm. And it remains for us to maintain the massage and the passive movements until he can regain power, and be able to replace our passive movements by active movements of his own. When he can do that, then the future treatment will rest with him, and so will the amount of success he gets subsequently. You may have noticed when he came in that to-day we are bandaging a small weight on to his arm, as that extends it. Then he will have massage, and probably the next day he will wear a sling with his arm up to his shoulder. By altering the position of his arm daily we hope to maintain the range of movement which he has got, and to increase it.

A Good Result after Division of Spinal Nerve-Roots.

The next patient, a little girl, is interesting for this reason: I do not suppose you have often had the opportunity of seeing one of the results of a very modern operation in cases which hitherto we could not benefit. She is æt. 7 years, and was an example of what is called cerebral spastic paraplegia. That is a condition in which you will find there is associated some mental deficiency. The spastic condition is confined to the leg; there is a spasm of all the muscles, causing a stiff straight knee, preventing the child from walking or getting about. And there is an adductor spasm, so that the legs are held close together, and they may even be crossed. So among the cases which we are trying to improve by surgery are children who in many cases are defective mentally, and who are unable

to get about. The pathology of the condition has not yet been worked out and is not properly understood. The idea at present is that the spasm in these muscles in the legs of the child is due to the fact that there is some source of irritation, which sends afferent impulses to the spinal cord, and so excites a reflex act, which gives rise to this spasm. Or, put in another way, the idea is that the spasm is dependent on the condition of the reflex arc. On that idea, an operation was devised on the posterior nerve-roots, those roots being divided. By the division of those roots one breaks the reflex arc of spasm, as it is called, and so the spasm passes away. On this child, on February 24th this year—4½ months ago—I did a laminectomy. The posterior roots of the second, third, fifth lumbar and second sacral nerves were divided. As you see, the child now, instead of having legs which were held out stiff so that she could not move them at all, is able to walk, although there is still some slight adductor spasm. You will notice the peculiarity of her gait, but she gets about quite well. She has that peculiarity for this reason: when we divided the posterior nerve-roots, we divided two consecutive nerve roots, not three. The result of such division teaches us a most extraordinary reversal of the physiology we were taught. There is no anæsthesia whatsoever. But if you divide three consecutive nerve-roots, you not only begin to get anæsthesia, but you also get trophic lesions. So we have learned that we can safely divide two consecutive posterior spinal nerve-roots but not three. The consequence of having to leave every third posterior root undivided is, as you see in this case, that the spastic condition is not completely cured. And so, while by means of this operation of dividing posterior nerve-roots we can improve these children immensely, we cannot get a perfect cure. There is one other point about this child, which you may have noticed when she walks, and that is, that there is some talipes left in both feet, and for this the child is at present staying in Great Ormond Street Hospital to have put right. After that is done she will be able to walk even better.

Paralytic Talipes.

This next patient, a boy, presents another example of talipes. This plate (skiagram) shows that the astragalus is missing in one place. He has a double paralytic talipes, and on both sides the astragalus has been removed. At the time this plate was taken the astragalus had been removed from only one side, and you can seen the result. The result is fair, but it is not so good in the right foot as it is in the left. He has come under care again to see if we can do anything to improve the condition of his feet. In order to bring these cases to a successful issue, it is necessary that you should work slowly; it is no good, in your treatment, proceeding by leaps and bounds, doing big operations at long intervals; but rather a number of small operations at short intervals—such operations as tenotomy or astragalectomy, or wrenching under an anæsthetic and putting the limb under plaster. By repeating this frequently, and perhaps putting the patient into some apparatus, one, so to speak, prunes the foot, so as to direct its growth in such a way as to slowly correct the deformity. It is by this constant care and these frequent operations that we get the best results in these cases of talipes.

Carcinoma of Nose cured by Non-operative Treatment.

Here is a girl, æt. 11 years, about whom, offhand, I am afraid I cannot tell you much. Some time ago she came up to Great Ormond Street Hospital on account of some condition about the nose, which, at that early stage, was considered to be a lupoid condition. On that supposition, it was treated by means of scraping and by the application of X rays for some time. Then it was noticed that this condition, which was imagined to be lupus, was increasing in amount, and forming a kind of tumour, and it was bleeding. A portion was therefore taken away and examined with the microscope in the first place by our own pathologist, Dr. Nabarro, and he came to the conclusion that this overgrowth, of which you can see a warty condition remaining, was a carcinoma. I have brought the case here in order to show you what is possibly a carcinoma in a child æt. 11 years. Our Great Ormond Street pathologist, recognising the unusual character of such a case, took the microscopic specimens up to the College of Surgeons, which I suppose we may regard as the highest authority, certainly in this land, on such a question. Mr. Shattock examined this specimen and came to the conclusion that the growth was

undoubtedly carcinoma. It was thereupon shown to various other pathologists in London, and their opinions asked. The unanimous opinion of pathologists was that it was carcinoma. That being so, we were plunged into a difficulty. If this growth was carcinoma, it was in a region which permitted of removal. But I am not fond of what may be termed "mutilating operations," and therefore I pointed out that if this were really carcinoma at that age, the likelihood of the disease having already affected the glands and the rest of the system was great, and hence removal of the primary growth would not do much beyond deferring the end. From those considerations, therefore, it was decided not to operate, but advice was given to fall back on such treatment as partial removal of part of the growth, applying X rays and radium. At the Radium Institute the application was combined with the administration of some decoction of alder, because it was found by the authorities at the Radium Institute that radium alone might be all right, but that when combined with alder the results were better. And as it was not likely to do any harm, we saw no reason why it should not be tried. The result is what you can see here. There is no longer a bleeding, ulcerating mass on the nose at all, but there is a warty condition remaining over part of the area, which the pathologists tell us is carcinoma. But, although they tell us it is carcinomatous, the condition, as I think you will agree, is not one which merits or justifies operation, and we shall continue the treatment, and I hope that it will prove to be all that is required. Of course, carcinomata do occur in the young, but not epitheliomata; that is to say, columnar-celled carcinomata, not squamous-celled ones. For instance, we know carcinomata to occur in the young, in the appendix and in the liver, but we do not know that epithelioma has ever been found in the child; in fact, I should say this child is one of the youngest in whom it has been found. So the case is not only interesting in that an epithelioma may appear in a child, but it is also interesting in that epithelioma in the young may appear to be eminently curable, or some of the cases may be, and curability by means short of operation—means such as we have pursued in this case. (Since showing the case at the Polyclinic all signs of disease have disappeared.)

Congenital Absence of the Fibula.

In this next case,* a boy, there is very little for you to see now, but I thought the case was worth bringing before you because the condition present—congenital absence of fibula—is easily recognised. In all the cases there are certain characters by which they are easily recognised. At birth the tibia is bent, and in almost every case there is a little raw surface on the convexity of the curve, so that when these cases are first brought to notice as examples of intra-uterine compound fracture of the tibia, the raw place on the surface of the bone being regarded as an indication of where the fracture was compound, it is that condition of so-called intra-uterine compound fracture of the tibia which should suggest to your mind the possibility of congenital absence of fibula, which, of course, can be proved by X-ray examination. Here are two pictures which will show what I mean. You can see a little scar where the fracture became compound. These skiagraphs show you the sound leg, and the unsound leg. In one of them you see the fibula is absent, and in another it is present. In these cases of so-called congenital absence of fibula, the congenital absence is not confined to the fibula, but, as is shown in that skiagram, you get congenital absence or deformity in what you may call the fibular segment of the leg—that is, in the fibula, tarsus and outer toes. I think this boy had only two toes; two or three outer toes were absent. Congenital absence of tibia is very much rarer, and it is not very likely to come to the notice of many of us. Congenital absence of fibula is much commoner than is usually supposed, and hence the cases are frequently overlooked, for the simple reason that people with this defect can walk and get on very well. When these cases do require treatment they prove very stubborn. Though this patient is only seven years of age, his leg had to be ablated because it was useless. It was useless in spite of the fact that since he was three months old he had been under skilled treatment; he had had everything done which could be thought of to try and preserve usefulness in the affected leg. But in spite of all that treatment, at seven years of age it was felt that it was of no use to try to do anything more, and so the case came to ablation. That is only too common—that

* See the 'British Journal of Surgery,' July, 1913.

these cases go on, and finally come to ablation. I think that could be avoided if only operative treatment were insisted on earlier. One of the greatest difficulties we have in these cases of congenital absence of fibula is that the articular surfaces which enter into the ankle-joint are irregular, so that although the tibia may be put straight, as in that boy, yet the surfaces of the ankle joint are irregular, and the ankle-joint is of no use. What I suggest is that operative measures should be instituted quite early, while the bones are actively growing, and that these measures should be as follows. The internal malleolus and external malleolus is replaced on either side by a plate and screws inserted into the tibia and astragalus, so that the growing astragalus and foot are held fixedly in a good position. If we did that I believe we could get regular surfaces in the ankle-joint, and at a later date it might be worth while to take out those screws and set the ankle-joint free again.

I think in this way it might be possible to avoid the very frequent occurrence of these cases, and their proceeding to amputation in spite of treatment.

Arthrectomy of the Knee.

This boy had arthrectomy performed for a tubercular knee. I have brought the case before you to illustrate an attempt which was made to improve the results. The ordinary incision which is made for arthrectomy of the knee is a horse-shoe-shaped incision from the inside, across the ligamentum patellæ, dividing the ligamentum patellæ, and the joint is opened in that way. Those patients suffer a good deal of pain, and they do so for two reasons: (1) the jumping brought about by spasmodic contractions of the quadriceps femoris, owing to the ligamentum patellæ having been divided. The other reason is the spasmodic contraction of the hamstrings, which occurs because their opponent in function, the extensor femoris, has been put out of action by the division of the ligamentum patellæ. Therefore one may use this incision, dividing the structures on the inner side of the patella and displacing the patella outwards. In that way one found one could diminish the pain considerably, and one also found that in doing the arthrectomy on the outer side of the joint, one was very liable to leave behind some of the disease which should have been removed. In fact, as you can feel, there is a certain amount of thickening about this knee which rather suggests that something more might have been done. So now I have taken to splitting the patella longitudinally, separating the fragments of the patella on either side, and then doing an arthrectomy, using the long median incision. I think that method is likely to be a useful one, and not only in arthrectomies, but in the case of foreign bodies in the patellar region, such as loose cartilages. As you know, injuries to the knee are very common, and they have an awkward habit of not getting well quickly. Operative treatment on a knee-joint is almost entirely confined to removal of a semilunar cartilage. It must be obvious to unprejudiced people that the internal semilunar cartilage is not the only thing in the knee-joint which gets injured. In fact if we were to argue theoretically one would point out that perhaps the internal semilunar cartilage, instead of being the first thing to be injured, should be about the last thing to be injured. And so an attempt was made to enable us to discover injuries to these other structures, other than semilunar cartilages, usually internal. For that purpose I have adopted in a number of instances the splitting of the patella longitudinally. This gives an excellent exposure of the joint. It enables you to see and to deal with lesions in the intercondylar space between the condyles, and with lesions of the knee-joint more completely than any incision hitherto employed. I find that most people bring forward an *à priori* objection to the incision, and that is, that whatever bone is divided, the convalescence must be a very long one, because the bone has got to unite again. In the case of longitudinally splitting the patella that is not true. In the first place, it does not lengthen the operation, because one does not have to wire the bone at all: you simply sew up the split tendon above the patella and the split ligament below the patella with a few stitches. Then you send the patient down to be skiagraphed. In four cases out of five, the report which you will get is, that there is no obvious lesion of the patella at all: the parts have come into such excellent apposition that the skiagrapher cannot demonstrate the fact which he knows beforehand—that you have sawn the patella through longitudinally. So one can dismiss from one's mind that the sawing through of the patella longitudinally makes the operation more severe, but it certainly makes it more efficient and it is much easier.

October 13th, 1913.

THE PATHOLOGY OF GROWTH. VOL. I: TUMOURS. By CHARLES POWELL WHITE, M.D., F.R.C.S., Special Lecturer in Pathology, Manchester University. Pp. 235. London: Constable & Co. Price 10*s.* 6*d.* net.

This, the first volume on Pathology of Growth, is assigned to tumours, and the allied subjects of hypertrophy and regeneration, etc. The variations in development, growth and functional activity, transplantation and grafting are lucidly explained in a readable and popular style. A most interesting chapter discusses the origin of tumours, their growth, and life-history. In the one dealing with their causation recent investigation is reviewed. In regard to the cause of cell proliferation, the author states that this condition in tumours is similar to cell proliferation under normal conditions. The assumption of a parasite to explain it, he says, is not only unnecessary, but makes an explanation of tumour growth more difficult. The statement merits quoting: "We come to the conclusion that the only direct stimulus to proliferation is the physiological stimulus of functional activity, or such physiological stimuli as act in anticipation of increased functional activity, and that extrinsic agents do not directly cause cell proliferation." Extrinsic factors, such as light, heat, chemical substances are separately considered. Intrinsic factors of tumour causation are offered chiefly by statistics. In discussing race and geographical distribution, some interesting features emerge. It seems that the Esquimaux is possibly the only race free from cancer, and that this disease is more common in temperate regions than in the tropics. In Europe it most frequently occurs in Switzerland, Denmark, France and Holland, least frequently in Servia and Hungary. It is more common in England than in Scotland, and less common in Ireland than in either. There is the curious circumstance that countries with a high cancer mortality have in general a low tuberculosis mortality and *vice-versâ*. In nearly all countries cancer is more common among females than among males. This is due to the frequency of tumours of the breast and uterus which are not comparable with the corresponding tumours in males. Omitting these, cancer, we are told, is more common in males than in females, and it is increasing at a greater rate in males.

The author has written an interesting book and one which cannot fail to be widely read. It represents the present stage of knowledge in this most difficult subject, and this knowledge ought to be the common property of the profession.

EPIDEMIC INFANTILE PARALYSIS. By Prof. PAUL RÖMER, Principal of the Institute of Hygiene and Experimental Medicine of Marburg. Translated by H. RIDLEY PRENTICE, M.B., M.R.C.P., late Resident Medical Officer to the National Hospital for Paralysed and Epileptic, Queen Square, London, Pp. 208. 57 illustrations. London: John Bale, Sons & Danielsson. 7*s.* 6*d.* net.

In view of the widespread interest consequent on the rapid results of investigation of this disease, the publication of this book is opportune. Dr. Prentice has done the work of translation excellently. The sporadic form, what might be termed the text-book variety, has of course been recognised for some time, but the new menace in the guise of epidemic infantile paralysis calls for ready recognition and prompt treatment on the lines of epidemic disease by constituted authorities. The book contains the results of Dr. Römer's experimental work, the communication and demonstration of the malady in monkeys. Strangely enough many of his postulates received confirmation in the recent report of the Swedish epidemic after the book was printed. On the Continent the malady is termed Heine-Medin disease after the two principal investigators — an unsatisfactory title, because when possible the name should express the essential character of the disease. Landry's paralysis would seem to be no longer a separate entity but infantile paralysis with a fatal issue. The book is not so much a text-book as a clinical treatise on this pest.

The prophylaxis follows that of the acute specifics, and the drug which up to the present has been of most use is urotropine. It alone or with serum represents the present stage of treatment. The book ends with a most extensive bibliography.

COPROSTASIS: ITS CAUSE, PREVENTION AND TREATMENT. By Sir JAMES SAWYER, formerly Professor of Pathology, also of Therapeutics in the Medical School, Birmingham. Birmingham: Cornish Bros. 2s. 6d. net.

Coprostasis is an old name for fæcal stagnation. The book is made up of six chapters from the author's well-known work, 'Contributions to Practical Medicine,' and is issued in this form so that it may more readily appeal to the general practitioner. The subject is dealt with exhaustively from all points of view. The author suggests after breakfast as the most suitable time for evacuation, except in those suffering from hæmorrhoids, in which case he thinks before bedtime is preferable. For drug treatment he relies chiefly on the socotrine aloes pill—aloes 1, 2 or 3 gr., sulphate of iron ¼ gr., and 1 gr. of hyoscyamus extract. This he gives at bedtime. If a saline cathartic is indicated he employs one or two teaspoonfuls or more of Rochelle salt an hour before breakfast dissolved in tea, coffee or cocoa. For piles he especially mentions the ung. ranunc ficar or pilewort ointment, which may also be exhibited as a suppository. The young practitioner will find many suggestions in the book for the treatment of a malady which, more perhaps than any other, engages his attention in general practice.

BANDAGING MADE EASY; by Miss HOSKINGS. SWEDISH EXERCISES; by Dr. LUKE. ESSENTIALS OF FEVER NURSING; by Dr. MAITLAND. HOW TO READ AND WRITE PRESCRIPTIONS; by Dr. MAITLAND. Scientific Press, Ltd. Imperial 32mo. Each 1s. net.

The above represent a series of hand-books which should prove popular.

The first fully illustrates the use of the roller and triangular bandage, each position having a picture to itself. For learners nothing could be better.

The second, on "Swedish exercises," as might be expected from the author, is an excellent little book, and, like the other, fully illustrated.

'Fever nursing' is readable and informing on the acute specifics. Some good suggestions are given both in regard to treatment and diet which even those who regard themselves as efficient nurses might well adopt.

The last book has been written in view of the National Health Insurance Act. The author is careful to explain how percentage solutions should be calculated, while all the terms of a prescription are dealt with separately. There is an extensive vocabulary, as well as a list of popular or alternative names for common drugs. A short chapter on incompatibility might fitly have been included.

THE STUDENT'S HANDBOOK OF GYNÆCOLOGY. By GEORGE ERNEST HERMAN, M.B., F.R.C.P., Consulting Obstetric Physician to the London Hospital. Second edition, revised by the author, with additions by R. Drummond Maxwell, M.D.Lond., F.R.C.S., Assistant Obstetric Physician to the London and Physician to Queen Charlotte Lying-in Hospital. 550 pp. London: Cassell & Co. 7s. 6d. net.

This handbook represents a condensed edition of Dr. Herman's 'Diseases of Women' and does it well. It is a most excellent book, and though primarily intended for the student, the practitioner will find it most helpful to dip into, or even for reference. There is a very good chapter on headaches, one on back pain and one on chronic abdominal pain. These serve as an efficient introduction to the subject pure and simple. The book is free from discursiveness, while the teaching is plainly and sometimes of necessity dogmatically put. The student cannot fail to find here a trustworthy guide to what (apart from children's diseases) will confront him as a general practitioner, and an extensive knowledge of which will also make him a successful one. There are about two hundred illustrations, some of which are in colour.

LABOUR ROOM CLINICS. By Capt. V. B. GREEN ARMYTAGE, M.D., I.M.S., Resident Surgeon, Eden Hospital, Calcutta. Calcutta: Thacker, Spink & Co. Pp. 67. 2s. 6d. net.

The lectures, of which there are twenty, embody the principles of obstetric teaching and practice at the above hospital. The teaching is terse and definite, on lines not unlike some of the well-known practical hand-books on midwifery. The book is interleaved, so that notes may be inserted when necessary for reference.

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A LECTURE

ON THE

CLINICAL AND PATHOLOGICAL RESULTS OF INFECTION OF THE FEMALE GENITALIA.*

By F. J. McCANN, M.D., F.R.C.P.,

Surgeon, Samaritan Free Hospital for Women.

Ladies and Gentlemen,—This evening I propose to tell you something about the effects produced on the female genital organs by infection. It is infection which is the cause of a large proportion of the diseases of women; and the study of infection has done more to make our knowledge exact in the domain of gynæcology in the past twenty-five years than, probably, anything else.

You know that the vulva, like the skin, is covered with bacteria. The normal virgin vagina is sterile; indeed, the vaginal secretion is credited with bactericidal powers. In a parous woman, especially one in whom the perinæum has been torn, or the vagina lacerated, the vagina is not sterile. But the cervical canal is sterile unless it be the seat of infection, and so are the uterine cavity and the Fallopian tubes.

The organisms which I intend to speak of to-night are, first of all, those which are grouped under venereal diseases. They are the gonococcus, the *Spirochæta pallida*, Ducrey's bacillus, and then the organisms which produce septic infection, which are streptococci, staphylococci, *Bacillus coli*, pneumococci. Then come tubercle bacilli in a special group. There are other, rarer organisms—gas-forming organisms, typhoid bacilli, diphtheria, tetanus, elephantiasis—but these I will only mention.

* Delivered at the Polyclinic.

Of the organisms which I have mentioned, the gonococcus is one of the most important causes of infection in the female genitals, and its results are seen either in the acute stage, or, more frequently, in the chronic stage. The acute stage is more often seen in venereal clinics, so much so that the statement has crept into some books that an acute stage is rare in the female. Of course, those of you who have experience of this department know at once that that is wrong. You will see, doubtless, on many occasions, examples of acute gonococcal infection. When a patient has sudden vaginal discharge, pain on passing water, and œdema of the labia, you may be certain she has got acute gonorrhœa. But it is the chronic stages which you meet with most frequently; and if you find infection of the cervix, infection of the glands of Bartolini, and infection of the urethra, that is to say, the three together, you may safely conclude that is gonococcal. No other infection will cause those three parts of the uro-genital tract to be affected. Then you get most frequently, of course, chronic gonococcal cervical discharge. And it is here that you meet with the chief infecting factor. *It is the chronic gonococcal cervical discharge which is the chief infecting factor.* It has been said that a gonococcal infection of the vagina practically does not occur.

That statement has been copied from book to book, and is due to the fact that the original experiments on the gonococcus and the observations on its action were made on the eyes of newborn children. It was there shown that the gonococcus had difficulty in penetrating the stratified corneal epithelium. And we know that in the vagina, whose walls are thickened, a vaginitis of gonococcal origin is extremely rare. But in the nulliparous vagina and in the vagina of children it is far from rare. To prove that that is so, a most interesting observation was made by a former member of this Polyclinic. I had done a complete hysterectomy on a patient, and that patient, some time after the operation, developed an acute vaginal discharge. It could not be a secondary infection from discharge coming from the cervix. Her doctor took some discharge from the vagina, and found the gonococcus, under the microscope, and also sent it to be cultivated. He had thus a complete and absolute proof of a primary gonococcal vaginitis. I do not know that that has been done before, but it gives you the proof complete, because the patient was devoid of a uterus, ovaries, and tubes, and had only the vagina left; hence there could be no question of infection from above. The gonococcus, however, exerts its ravages higher up in the genital tract, and the chief cause of an ascending infection is the occurrence of pregnancy. Apart from pregnancy, other causes are the use of the syringe, or sexual intercourse during the existence of the gonococcal cervical discharge. In consequence there may be acute metritis or acute salpingitis, which subsequently dies down, and a chronic salpingitic change remains. The affection in the body of the uterus tends to proceed to a natural cure, whereas in the tubes the tendency is for those tubes to become adherent, and their abdominal ends to be sealed; and you may later find an accumulation in the tubes, either of a serous discharge, forming a hydrosalpinx, or a purulent collection forming a pyosalpinx. In many cases you have a mixed infection. If you have a pure gonococcal infection, what happens is that you get an adhesive peritonitis without the formation of pus. Remember, the gonococcus is not a pus-producing organism. Its effect is so to damage the mucous membrane on which it is implanted that pus organisms grow and develop in that situation. The great difficulty in connection with cultivating the gonococcus is that it soon gets overgrown by pus organisms. When you get a pure gonococcal infection infecting the peritoneum and tubes, you simply find an adhesive peritonitis, without pus. That adhesive peritonitis causes absolute sterility; that is one of the most frequent consequences of ascending gonococcal infection: either the patient never conceives, or where the infection has spread up the tubes in consequence of confinement she may have what has been called a "one-child sterility," but which I prefer to call "one-child fertility." In other words, she is sterile after the birth of that particular child. These cases are associated with much chronic ill-health, with menstrual pain and backache, and evidenced clinically by the discovery of thickened masses in the region of the tubes, often causing the uterus to be pulled backwards, and fixed more or less firmly in the pelvis. Such a clinical picture I daresay you are all familiar with, and it is what you see as the result of ascending gonococcal infection. Remember that the gonococcus has a tendency to remain

in certain parts of the female genital organs in the same way that it does in the male. And those situations in which it tends to persist are the Fallopian tubes, the cervix, the Bartolinian glands, and the urethra.

Now, coming to the *Spirochæta pallida*, which, as you know, is believed to be the organism causing syphilis, you see syphilitic infection in the female genitals in three stages: primary, secondary and tertiary. It is an extraordinary thing how the statement that a primary sore is rarely seen in a woman has crept into text-books and been reproduced from one book to another. During the last twenty years I have never had sent to me a correctly diagnosed primary sore in a woman. I have had several cases sent as something else when the cause was a primary sore. Three or four of them were sent as cases of cancer, others as simple dyspareunia, others as women with a discharge, and so on. Why have these cases not been correctly diagnosed? The reason is that one of the most common situations for a primary sore in a woman is the fourchette. The fourchette being in the middle line of the body, that situation is relatively avascular. Where there is a primary sore there is little or no induration. It looks like a flattened ulcer with a well-marked red margin and slightly grey base; and when you catch it between the finger and the thumb you will find only a certain parchment-like induration, not like what you find in true Hunterian chancre. Sores are common in that situation and on the labia, and they are often overlooked. They are frequently the means of infecting the finger of the examiner, especially when the sore is at the fourchette. You find sores on the labia with all the definite characteristics of the Hunterian sore. And not only so, but you frequently get well-marked induration, so that the whole labia may be indurated. Therefore if you find a sore and unilateral induration of the labium you should always suspect syphilis. And not only do you get primary sores, but multiple primary sores, sores on opposite sides of the labia. Then you find the secondary manifestations, mucous patches, and condylomata, which are pretty obvious and do not lead to mistakes in diagnosis. Then there are tertiary manifestations. Only the other day, on reading a certain book, I saw the statement that tertiary manifestations are rarely seen. This is incorrect. I have seen any number of them. I have seen, in a young woman, the two labia absolutely excavated by gummata. I have seen, in a woman æt. 70 years, a gumma which had made a horseshoe-shaped ulcer extending from one labium to the other, the patient having been sent for treatment as a case of cancer. So gummata occur on the labia, and you must not mistake them for cancer, because they readily yield to treatment. There is some evidence to show that syphilis exerts influence on the genital organs higher up. There have been described cases of salpingitis due to syphilis. It is difficult to prove, and most of them are probably mixed infections. But I think it causes chronic metritis, associated with free menstrual, and sometimes with intermenstrual, loss. Such cases I have found to be benefited by iodide of potassium, and the same is true of certain congenital syphilitics. I have arrested hæmorrhage with iodide of potassium in patients the subjects of congenital syphilis. So it is a point to bear in mind where a patient has previously had syphilis that metritis may occur in consequence of it. But where there have been repeated miscarriages there is a chance of there being mixed infection, which is such a frequent sequel.

Ducrey's bacillus is characteristic of a soft sore, and soft sores on the female genitals are much the same as those in the male, and those cases are frequently associated with enlargement of the inguinal glands, which goes on to suppuration, that enlargement being different from the shotty hardness associated with primary sore.

Now, with regard to septic infection: You have first of all to consider the effects of the streptococci, then of staphylococci, then of *Bacillus coli*; and lastly I shall refer to the pneumococcus. These infections may occur after operations, after full-time labour, or abortion; or they may complicate new growths, or be found on the external genitals.

First, with regard to these infections occurring after operations, I need not detain you by going into the well-known fact that septic trouble readily follows unclean operations. Besides, you may find acute metritis following curettage for gonorrhœa, or following on curetting for gonorrhœa, acute salpingitis. Therefore do not curette a gonococcal metritis. Sometimes perforation with peritonitis is produced. That is an accident which should be avoided. But you meet with the

chief examples of infection in association with abortion or full-time labour. And in these cases which you meet with the infections are either very acute or are of a subacute nature. Let us consider the latter of these first. One of the most common results which you see clinically from a mild infection is chronic metritis. Amongst the poor there are numerous examples of the slighter forms of infection following upon a miscarriage; and these are evidenced clinically by a chronic metritis, chronic enlargement of the uterus, associated with free loss at the monthly periods, with intermenstrual discharge, and probably backache and bearing-down. Those cases you must have met with fairly often. Another common affection is the result of a slight cellulitis. You know that one of the commonest injuries of child-birth is a tear of the cervix, that tear being commonest on the left side. If that tear gets infected, as it frequently does, the patient gets a cellulitic deposit in the left broad ligament. Usually those cases are seen in patients who get about and do their ordinary work, and, as a matter of fact, the cellulitis disappears. But what happens is, that an indurated cicatrix forms in the left broad ligament, which pulls the cervix towards that side, and that cicatrix remains a tender spot for a long time, often for years. The third commonest type of mild infection here is chronic cervical discharge. Those are the three conditions which you meet with every day: chronic metritis, cervical discharge, and healed cellulitis. In addition you get effects produced in the organs higher up; you get salpingitis and ovaritis. These may be evidenced by acute symptoms if there are acute infections; but they are more commonly associated with subacute symptoms; a chronic infection is what you mostly find. And in these cases the infection is evidenced clinically by the existence of swellings in the position of one or other tube felt behind the uterus, and often dragging the uterus backwards, as in the gonococcal cases. Those infections due to streptococci and staphylococci tend to get well. A woman may have such an infection of the tubes and yet the tubes may recover perfectly, and she may be able to bear children, which is the test of complete recovery. It is otherwise with gonococcal cases. A gonococcal infection tends to produce adhesion and closure of the abdominal ends of the tubes, and so permanent sterility.

There may, however, be a mixed infection—gonococcal, staphylococcal, and streptococcal; and in these cases, too, you find the predominant effect of the gonococcus frequently manifest.

Another result of infection following miscarriage or confinement is thrombo-phlebitis. You often find a patient who has been getting on fairly well after a confinement, suddenly develops a temperature, and the temperature keeps up. You examine, but you cannot find anything wrong. She then begins to get swelling of the left leg. In addition to the thrombo-phlebitis you find there is a blocking of lymphatics. That blocking of the lymphatics causes a swelling of the leg, which leads secondarily to blocking of veins. It is the mixing up of those two types which has led to so much bad treatment of these conditions. You would be surprised to know how many women are permanently lamed on account of these swollen legs following upon confinement. The reason is that it seems to be fixed in the medical mind that they are mostly thrombophlebitic, and that these legs should not be touched or moved; whereas, as a matter of fact, a large number of them are lymphatic, with pressure on the vein as a secondary consequence; and they require to be treated by massage and movement I hope, on another occasion, to go into the treatment of those various infections; at present I am speaking of the clinical manifestations and pathological results. But I feel strongly on this question of the swelling of legs after confinement, because I have seen so many people lamed for months or years, owing to their not having been treated properly for it.

There may be acute general peritonitis in the cases where there is very acute infection. There may be infection in association with new growths. Cancers may become virulently infective, and sometimes the discharge from these growths contains streptococci. That is why, after some operations for cancer, the patient dies in from twenty-four to forty-eight hours, of acute septicæmia. Streptococci have been found, in such cases, in the peritoneal cavity; they have been found in the discharge from the cancer. It is therefore very important, in the examination of cancer cases, not to infect them; you should wear rubber gloves or finger-stalls when examining.

Polypi frequently get infected, and there may be chronic, or even acute, poisoning as a consequence.

Infection is seen from organisms, especially staphylococci, on the external genitals. Some women suffer from crops of boils, which are mutually contagious. Then the Bartolinian cysts may become infected. Do not condemn everybody who has a suppurating Bartolinian cyst as a subject of gonorrhœa. Frequently those cases are staphylococcal infections. The gonococcus, however, does not infrequently infect a cyst, and there may be suppuration. It is gonococcal infection *plus* another infection. But there may be an infection by staphylococci without any evidence of gonococcal infection. You have probably all read that most cases of vulvo-vaginitis in young children are said to be gonococcal. That is absolutely wrong; a large number of them are staphylococcal in origin. I have taken the trouble to examine the pus and make cultivations. A certain proportion are undoubtedly gonococcal, as everybody knows; but, on the other hand, a number are not. So please do not condemn a child with vulvo-vaginitis as suffering from gonococcal infection unless or until you have isolated the gonococcus and cultivated it.

We now come to a consideration of the *Bacillus coli* infections. Why the female organs are not more frequently infected with the *Bacillus coli* I cannot understand. There must be a wonderful protective mechanism. We know when the perinæum is torn, and the vulva is gaping, that there is frequently *Bacillus coli* infection. And you can generally recognise an incompetent nurse from the way in which she sponges the vulva. An incompetent nurse sponges from the anus towards the vulva, and thus tends to wash the *Bacillus coli* into the vulva. Nurses should, of course, always be taught to sponge in the opposite direction. As I say, why infection by *Bacillus coli* does not oftener occur I fail to understand. We know that women can get infected with tears of the perinæum, and also where there is a markedly gaping vulva; and we know also that *Bacillus coli* cystitis is now proved to be quite of common occurrence; and remember that it occurs with acid urine. Moreover, the *Bacillus coli* attacks the genital organs frequently from the bowel, infection spreading to the right tube and ovary from the cæcum and appendix. In the case of infection from the sigmoid, it spreads to the left tube and ovary. In these cases there may be the formation of an abscess on the left side, between the sigmoid colon and the pelvic colon and the appendages. But you see more frequently still the effects of *Bacillus coli* in the adhesion of intestine to tumours, and to the tubes when they contain pus; and also in the case of tubal gestation. A well-marked example is seen where there is a cyst with its pedicle twisted, and the bowel becoming adherent to the cyst, there is a very virulent infection of that cyst; so virulent that if the contents escape into the peritoneum the patient will probably die of acute septic poisoning.

The pneumococcus I only mention as an interesting bacteriological curiosity. It is not commonly found, and would be only discovered by special methods of cultivation.

But I must, in conclusion, say something about tuberculosis in connection with the female genitals. That is one of the most interesting subjects you could possibly study. I did not know it was so common until I had occasion to open the abdomen frequently. It exerts a more destructive influence on the female genitals than does any other disease. I have seen complete destruction of the tubes and ovaries as a result of tubercle. That was in the case of a woman who came to the hospital with a primary prolapse—the uterus came down through the vulva. She was single, and she had never menstruated since her periods first began at fifteen years of age. They occurred only once or twice, and then stopped permanently. At that time she had an attack of what must have been tuberculous peritonitis; she had well-marked tuberculous scars on both sides of the neck. I opened the abdomen for the purpose of slinging up the uterus, and I then found that the peritoneum of the rectum passed on to the peritoneum of the bladder. One could see no uterus, no tubes, no ovaries. I accordingly incised between bladder and rectum to get down to the uterus, and I found the Fallopian tubes represented by fibrous cords, and the ovaries were represented only by small fibrous nodules; in fact the tubercle had completely destroyed the tubes and ovaries. That is worth bearing in mind, that the disease can do such damage, in cases of phthisis in young people. You will be told that such young women never menstruated again. Although the disease may not completely destroy ovaries and tubes, it may partially destroy them; and although the patient may menstruate, she cannot bear children. Hence it is found that tubercle is

a not uncommon cause of sterility. The tubes get sealed up by tubercle, in the same way as I have mentioned they become sealed up by gonorrhœa. Still, it is only recently that it has been realised what a common cause of sterility tubercle is; there is old-standing abdominal tubercle which has healed, and as a result of the healing process the tubes have become sealed for all time. The question is, How does tubercle get to the genital organs? There may be a primary infection. It has been proved that a woman may be primarily infected through the spermatozoa. A few definitely authenticated cases of that have been published. However, the bacilli may enter through the respiratory mucosa, or through the mucosa of the bowel, and settle on the least resisting portion of the female genitals; and that area of lessened resistance is often supplied by Fallopian tubes which have recently been the seat of gonococcal inflammation; so that gonococci and tubercle bacilli have been demonstrated together in the pus of a pyosalpinx. The patient gets tubercle bacilli from the respiratory tract, or from the alimentary tract, and those organisms settle on the weakened tissue and the result is a tuberculous salpingitis. Or the organisms may gain entry by a small wound and be carried by lymphatics. But the commonest cause is a secondary infection, a hæmatogenous infection—in other words, an infection carried through the blood, the primary focus being in the lungs or the bronchial glands.

The next most frequent form is infection spreading from the peritoneum directly to the tubes. It is so common to find a tuberculous peritonitis with the tubes also infected. Infection may spread from the bowel, from the urinary tract, or from the uterus and vagina. The clinical manifestations of the ravages of tubercle in the female are, the occurrence of sterility, and sometimes, as in the exceptional cases which I have cited to you, the occurrence of amenorrhœa. But what is much more frequent is the occurrence of uterine discharge. If you are treating a delicate young patient who has uterine discharge, bear in mind the possibility that she has tubercle, and do not curette her. If you take the trouble you can sometimes demonstrate the tubercle bacillus in the discharge. Also in the cases where the peritoneum is infected, you find this evidenced clinically by the peculiar swelling and dough-like consistence of the abdomen, the irregular diarrhœa, the dyspeptic phenomena, and often the wasting. Or there may be a definite abscess formation between the coils of intestine. These are the cases in which the peritoneum is infected, and the infection has spread to the tubes. But the point which I wish to make most emphatically here is the occurrence of sterility from tubercle, and the extraordinarily destructive effects tubercle may produce on the tubes and ovaries.

The other organisms which I just mentioned are so rare that they are not of much practical importance; those which I have dealt with are most frequently met with in ordinary every-day practice.

October 20th, 1913.

THE ÆTIOLOGY OF ENDEMIC GOITRE: THE MILROY LECTURES. By ROBERT MCCARRISON, M.D., R.U.I., M.R.C.P., Major, Indian Medical Service. London: John Bale, Sons & Danielsson, Ltd. Pp. 216. Price 10*s.* 6*d.* net.

In his quest for the source of endemic goitre, Major McCarrison has exhausted a most extensive bibliography and visited many climes. Endemic goitre unfortunately enjoys a world-wide reputation and cause, though it displays an obvious partiality for certain districts. Thus, in parts of Italy it affects 1½ to 3 per cent. of the population. It is almost constantly associated with mountainous regions, the notable exceptions being Norway and the Highlands of Scotland. The disease exhibits a distinct seasonal prevalence, favouring May, June and July; females, more than males, are liable to it. Certain salts, such as magnesia or lime, were at first invoked as causal agents, but it would appear that the *contagium vivum* exists in the water and also the soil. The habitat in man is in the first instance the intestinal tract. Treatment at present aims at intestinal antisepsis. The author favours thymol in 10-grain doses twice daily, also lactic acid bacillus and vaccines. Such treatment is applicable only in cases of recent origin, when cystic and adenomatous changes have not supervened. A map indicating the approximate distribution of endemic goitre over the globe completes an interesting volume.

CLINICAL LECTURE
ON
THE PULMONARY TRIAD: BRONCHITIS, ASTHMA AND EMPHYSEMA.

At the Royal Victoria Hospital, Belfast.

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THE case which we have to-day is apt to be treated by the student with some superciliousness; it is termed an "old bronchitic," and the treatment is often summed up in the stock bottle, labelled—"Mist. Pect. Stim." But whether we consider the annual mortality to the nation—for many thousands die each year from this affection—the importance of the affection to the patient and to his family, or the pathological problems, both old and new, that are involved, we have a disease of great consequence and of perennial interest.

The notes are shortly as follows: He is a male, æt. 54 years. His family history shows that his mother and a maternal aunt suffered from chronic bronchitis, that one of his mother's brothers and one sister died from "consumption of the lungs," that one of his own brothers died young of some chest affection, and that one of his children has already had some "bronchitis," which attacks the child suddenly; he says that there is a "weak chest" in his mother's family. It might be well to form a clear idea of the significance of this popular term. A "weak chest" means one of two things: either that there is a tendency in a person to pulmonary tuberculosis, or that there is a tendency to what is popularly called "asthmatic bronchitis." Those who suffer from the latter proclivity are also perhaps more subject to tubercle in the lung, but we find a large class in whom "phthisis" is a family affection, and another class in whom "asthmatic bronchitis" is a family affection, without proclivity to the other disease. In his personal history you learn that till some seven or eight years ago he had enjoyed fair health, that since then he has had a winter cough, that these seasonal attacks have become longer and longer and of greater severity, that he is liable to be awakened at night with great shortness of breath, that cough and expectoration continue during the next few days, and that for the last two years he has had to give up his occupation, which is that of an iron-moulder, during the winter. During these attacks the paroxysms of coughing are severe, there is a large quantity of expectoration; even in summer now he is never quite free.

We now come to the personal examination: it is necessary in dealing with patients to follow a routine, and to be consistent and exact in the use of your terms. A patient tells you certain symptoms of which he complains: you render this information more full and precise, and you elicit further facts by questioning; this is called the "oral examination"; it is of great importance, and if skilfully and patiently carried out may afford data sufficient in itself on which to base the diagnosis; but in a great majority of cases the "physical examination" is of equal or greater importance. Just as there is in the dissecting room a definite routine laid down for the examination of the various systems of the body, so clinically there is a routine at the bedside, by following which you make your examination of the various systems of the body in the living patient. This physical examination is first carried out at the bedside, and then subsequently completed in the clinical laboratory. Having ascertained all the facts you can, you now make your "diagnosis": this is a process of reasoning on all the facts you have obtained by "examination." You must not be content to tack a label on one organ of the patient; you must estimate his "constitution," that is to say, the condition of his arteries, of his nervous system, of his susceptibility or his powers of resistance to various infections; but also you must ascertain the condition of the individual organ or system which is more immediately at fault, or the exact nature of the infection, if such exists. You must translate your signs and symptoms into pathological terms.

Going through the routine of the clinical examination of the lungs, which is clearly the system involved, we notice on inspection that the chest is somewhat barrel-shaped, his shoulders are raised, the substernal angle is obtuse, the ribs approach the horizontal and there is slight upper dorsal kyphosis; the angle of Louis in the sternum is well

marked; the sterno-mastoid tendons are rather prominent, and there is little respiratory excursion although much respiratory effort. The patient's face is very faintly cyanosed, or what one might term slightly dusky or "blae" coloured; his lips have a duskier red than normal. On percussion of the chest we find a full resonant note present both back and front; on the left side we get practically no area of superficial cardiac dulness; the very inner or sternal margin of the fifth interspace has on very light percussion, not a dull, but a *very slightly* resonant note which is masked by the sound due to the closeness of the cartilages. On the right side, we know that in the nipple line the fourth and fifth interspace should begin to lose their full resonance, and that the sixth space should on light percussion be dull; but here we find that there is little change in the fourth and fifth space, and that resonance extends to the sixth and even seventh space, so that we have only about an inch and a half to two inches of superficial hepatic dulness, instead of four and a half, which is the normal; this increased area of pulmonary resonance extends into the axillæ, and, slightly diminishing, round to the back on both sides. Vocal fremitus is unchanged. On auscultation we find a short inspiratory murmur, a prolonged rather low-pitched and, in places, wheezy expiratory; the intensity of the respiratory sounds is less than normal and not at all in keeping with the rather forcible respiratory efforts; there is no difficulty in inspiration, there is a prolonged difficulty in expiration; we get "adventitious sounds" also, some *râles* more marked at bases, and some rather indefinite rhonchi. Vocal resonance is perhaps somewhat diminished.

In auscultating the lungs, always pay heed to the ordinary respiratory sounds and their modifications. You should become familiar with the normal or healthy sounds; your stethoscope should be for some months at your own bedside so that you can practise on yourself, and your ear should know the sound found over your own trachea, sterno-clavicular area and axillæ; and on the apex, the inter-scapular areas and base of some friend. You can carry this procedure out at your leisure, repeat it as often as you please, and at the same time experience the unpleasant sensation of fulness of head and giddiness due to repeated forced respiration, which a student not infrequently forces on a weak patient from want of practical knowledge.

This "tubular" (or better "tracheal"), "bronchial" and "vesicular" breathing is modified by disease. "Bronchial" breathing in a "vesicular" area means consolidation of the lung, and the conduction of the "bronchial" sound to the stethoscope; it varies in degree, and if it arises from a superficial small bronchus, it is high-pitched and "whiffing." If a cavity in consolidated tissue is present, we find the "bronchial" breathing further modified; it has a cavernous, hollow, or metallic character, such as is produced by blowing over the neck of an empty bottle.

Adventitious sounds can be classified as follows. We have first of all sounds produced by the movement of the stethoscope on the chest-wall, the rubbing together of the rubber tubes, the rustling of hair, and other outside causes: a little practice soon enables us to distinguish these. More difficult and more important are the sounds produced by the contraction of muscle, which is of an indefinite rumbling character, and is especially apt to mask the respiratory murmur when the patient is shivering. Occasionally, also, we hear sounds near the shoulder-joints, which are apt to be mistaken for intra-pulmonary; they are probably due to a local fibrositis. Thirdly, we have pleural sounds: it is not uncommonly thought by the beginner that there is only one pleural friction sound, whereas there are several: (*a*) The early shuffling friction, generally heard in the first stages of pleurisy, which can be rather poorly imitated by putting the back of one hand against the ear, and rubbing, in rather a jerky manner, the dry palm with the dry palm of the other. (*b*) The creaking or dry shoe-leather sound, more commonly heard in chronic thickened pleurisies. (*c*) A modification of this type is a "crumpling" or "crunching" sound, not unlike the sound produced by walking in snow. (*d*) The *râle*-like sound, which varies from a short, soft, unmusical sound to a large, soft, unmusical, resembling the sound made by separating two pats of butter; it and (*c*) are heard best on the subsidence of pleural effusion, but may be obtained wherever pleural deposit is soft and sticky. This latter sound is at times very confusing and is often thought to be due to fluid in the bronchi, and called a *râle*.

The last section of these adventitious sounds embraces the intra-pulmonary kinds; they are divided into (*a*) rhonchi, which are musical and continuous, and if high-pitched are called sibilant,

if low-pitched are called sonorous. They are due to local tumescence of the mucous membrane of the bronchus, or to collections of thick stringy mucus, which stretch across the bronchus, or partially block it. (*b*) *Râles:* These are interrupted, unmusical sounds; they may be modified in two different ways: (1) The sound may be very small or very fine, or it may be medium or it may be large and coarse, so that we may have a very fine, a fine, a medium, a coarse, a very coarse *râle*. (2) If the *râle* is produced in unchanged lung-tissue, it has a soft character; if, on the other hand, it is produced in consolidated lung-tissue, it has a sharp, clear, high-pitched, well-defined character; these two sounds have been compared to the sounds produced by blocks of wood on the one hand, and by hard stones on the other, rattling together. (3) Lastly, if a *râle* is produced in a large smooth-walled cavity, it has the sharp, clear definition of No. 2, but in addition has a peculiar, metallic, hollow or cavernous ring about it. (4) A soft rustling *râle*-like sound is heard over distended parts in emphysema. (*c*) The third sound is called "crepitation." It is always necessary in science to be exact in the use of terms; consequently I have discarded the employment of such expressions as "dry sub-crepitant *râles*," etc., as they really convey no clear image to one's auditory consciousness; these terms are usually applied to fine, sharp *râles*, but they are mostly unintelligible and vague and convey different impressions to different ears. I give the surgeons a present of the term "crepitus," to label the sound and feeling produced by the grating of the ends of a broken bone, or of an osteo-arthritic joint. I limit the use of the term "crepitation" to the peculiar, extremely fine, explosive sound, heard in the first stage of pneumonia; it differs not merely in degree but in kind from the very fine *râle* of capillary bronchitis; it is still doubtful whether it is due to the separation of the sticky walls of the air alveoli during inspiration, or to the very earliest stage of pleural inflammation, and so antecedent to the dry pleural rub or shuffle, already described; it resembles the sound produced by the sudden relaxation of the pressed fingers on an indiarubber sponge. Now let us return to our case; we have soft fine and medium *râles*, and have both sibilant and sonorous rhonchi; these mean muco-pus in the bronchi.

The clinical examination of the nasal passages, of the pharynx and of the larynx and trachea in this case does not reveal any abnormality. The sputum you see is copious; we have 4 oz. of a muco-purulent expectoration in twelve hours; some of this has been spread on a slide and the film has been stained and examined for tubercle, but none of the bacilli of tuberculosis have been found. There are, however, large masses of cocci, both small and large; here and there also, diplococci with a slight envelope are noticed; there are also bacilli of numerous kinds and streptococci, and some larger bodies, evidently yeast-cells, and a large number of cells with nuclei. Now a number of these bacteria are found in the mouth and throat, and so to eliminate this fallacy the patient is provided with a glass of hot water in the morning before his early paroxysm of coughing, with which he repeatedly rinses his mouth and gargles his throat; this induces cough and he now expectorates into a wide-mouthed bottle, which with cork has been boiled, and the bottle recorked. As soon as possible, some of the sputum is put into an ordinary small strainer which has been rendered sterile by boiling or holding in a Bunsen's flame, and the ordinary tap-water allowed to flow roughly over it; by this means the outer contamination of the sputum from pharynx or mouth is washed away, and as the tap-water does not contain any pathogenic bacteria of the respiratory tract, it may be considered as sterile for this purpose; this, I believe, is Armstrong's method of St. Mary's. To ascertain the nature of this bacteriological flora, we must further proceed to make various cultures, of which the details, however, are outside our immediate object; it will suffice to state, however, that the chief organisms found in these cases are pneumococci, the *Micrococcus catarrhalis*, staphylococci, streptococci, the bacillus of Friedländer, and occasionally the *Micrococcus tetragenus* and the influenza bacillus. The importance of this knowledge and that of the relative proportion of each microbe will be referred to again when we are dealing with the treatment.

A point of considerable import revealed on general observation of the chest is that there is well-marked epigastric pulsation, but no apex-beat visible. This pulsation is close under the left costal cartilages, and when we think of the position of the right ventricle lying on the diaphragm in this

region, we see that the pulsation is due to the right ventricle and points to its enlargement. The apex is displaced from the chest-wall by the encroachment of the hypertrophied right ventricle, and is certainly deeply covered by the emphysematous lung. Remember that a cardiograph of the right ventricle will be an inverted one when compared with that obtained over the apex-beat. The apex projects out, the walls of the ventricles draw in, during systole. The liver is a finger's breadth lower than normal, there is no albumen in the urine, and no dropsy of the legs nor of the abdomen; but there was a trace of albumen when the patient was admitted, and a slight pitting about the ankles; both disappeared in a few days.

Adopting the method of exclusion in working out our diagnosis, we can say at once that it is not an affection of the nervous, abdominal, or locomotor systems. The heart is acting regularly, the apex-beat as far as we can judge by auscultation is in its normal position, or just a little out; there is no sign of hypertrophy of the left ventricle, and the sounds are clear, although of low intensity from feeble conduction due to the overlying lung, with the exception of a slight basic systolic murmur, which is so common as in most cases not to be pathological. We do find, however, a history of serious pulmonary trouble, hereditary in the family, of personal liability in the patient, and the physical signs in the lungs, so it is perfectly legitimate to arrive at the conclusion that the main and primary trouble is pulmonary. What form of lung trouble presents the history and physical signs we have just learnt? We can at once say that we have not acute lobar pneumonia, nor advanced pulmonary tuberculosis, nor pleural effusion. We cannot, however, get rid of "chronic bronchitis," that is a chronic catarrhal condition of the mucous membrane lining the bronchi, nor of "asthma," nor of "emphysema," with the same facility. In fact, on the contrary, we have evidence of the presence of all three; we have paroxysms of cough, *râles* and rhonchi on auscultation, and copious muco-purulent expectoration, which we have proved comes from the bronchi—all strong testimony to the existence of chronic bronchitis. We have sudden onset of attacks of dyspnœa, coming on in the early morning, eased by the administration of anti-spasmodics, slowly lessening and allowing sleep in a few hours, but followed during the next few days by increased signs of catarrh, all proving the existence of a certain amount of asthma or spasm of the involuntary muscular fibres of the bronchi. And lastly, we have the slight duskiness of colour, the typical aspect, the barrel shape of chest, the spread of pulmonary resonance over the area of superficial cardiac dulness on the left, and of the hepatic dulness on the right, and all round at the base on both sides, with a faint respiratory murmur and at places a soft rustling sound heard in these regions, all pointing to emphysema or loss of elasticity in the lung-tissue, the running together of air-sacs, the formation of large air-bullæ and the intrusion of the lung down into the complementary pleural sinus, obliterating the superficial cardiac and hepatic dulness. This cannot be due to pneumothorax, as it is symmetrical, which pneumothorax could not be; the respiratory murmur is heard, although faint, while in pneumothorax it would be absent, or bronchial over a bronchopleural fistula.

Our text-books on medicine describe three affections—bronchitis, asthma and emphysema; but we frequently find this type of case where all three conditions are present in varying proportions. You seldom get a case of chronic bronchitis without emphysema, and there is also, not infrequently, an asthmatic element, sometimes more marked, sometimes less. This is shown in the popular term "asthmatic bronchitis" or "bronchial asthma," where "bronchial" means either "bronchitic," or is used in contra-distinction to "cardiac asthma," "uræmic asthma." Likewise most cases of bronchial asthma eventually develop bronchitis and inevitably develop emphysema. We practically never get a case of primary emphysema; the elastic tissue of the lung may be poor stuff from birth, and so some lungs yield more easily than others as some arteries harden more easily. The two chief *diseases* that we apparently have are: (1) This microbic catarrh of the mucous membrane of the bronchi, to which some people seem much more liable than others. It is both a question of exposure to infection, but also and by far the more important, of resisting power in the individual. How far this resisting power is local in the lungs, how far general or constitutional, has not been determined. We are also on the trail of the infection, but we have not yet caught our quarry; we cannot yet assign the values to each microbe. (2) The second disease is the

paroxymal attacks of spasm of the bronchial muscles, which may be excited by certain inhalations, by chills to the external surface, by internal toxins of great variety from food and other causes, but probably the most frequent cause, by onsets of microbe-caused catarrh of the bronchi, which is the case in this patient. Emphysema is a *condition*, not a disease, and is practically always secondary to one of these two diseases. We have, then, this type of case; it is really not three diseases, but one disease; it occasionally runs in families and shows itself, sometimes in youth, generally before adult age; it is one of the forms of "a weak chest," it is very common in those whose occupation involves the inhalation of much dust; it is much less frequent among "hacklers" in Belfast, since the introduction of the new moist system of ventilation.

Prognosis.—The prognosis in these cases is on the whole not good; it depends greatly, however, on the social position and wealth of the patient. If the patient is of the working class and is condemned to live in unhealthy surroundings, in small, overcrowded, ill-ventilated, ill-heated rooms, to work at an unhealthy occupation for the support of his family when he should be confined to bed, to be ever exposed to the temptation to try and compensate for want of appetite and unsuitable food by frequent resource to alcohol, an early pneumonia or a capillary bronchitis may relieve him of both life and suffering. If, however, the circumstances are not so sordid, the emphysema tends to progress; this throws a strain on the right ventricle, which at first hypertrophies, but finally yields and dilates; the liver, stomach and intestines become congested, and digestion and metabolism are interfered with, albumen appears in the urine, and œdema in the legs; then ascites, hydrothorax, and lung œdema end the scene. If, on the other hand, the patient is in good circumstances, and able to seek a warm climate during the winter, or even to make an artificial climate in a series of warm, well-ventilated rooms in his own house, where he is confined during the cold winter and spring months, it is surprising to what an age such patients may live, and what comfort they may enjoy the other seven months of the year. Of evil significance are a bad family history, rapid development of the emphysema, repeated attacks of bronchitis, especially if the asthmatic element is marked, and early dilatation of the right ventricle.

Treatment.

We divide treatment into two sections:

Section 1: *Preventive.*—Is there any method of anticipating the onset of this disease? The science of eugenics may perhaps in the future tell us how to obtain healthy and strong elastic tissue in our lungs, how to avoid a "weak" chest; at present such a discussion would be purely academic. If, however, a member of a family in which this form of weak chest is rather frequent, consult us, can we do anything to anticipate the onset of the bronchitis, the asthma and the emphysema? Such an individual should undoubtedly lead an open-air, healthy life; he should be lightly clad with underclothing of either linen, silk or open-meshed cotton to avoid over-heating of the skin, and so to "harden" it, *i. e.* to be less sensitive to the hundred and one chills we are all exposed to. He should be put to some open-air occupation, preferably in a suitable colony, like New Zealand or South Africa; on no account should he be apprenticed to confined office-work, to foundry or mill-work or to similar occupations. He should avoid crowded assemblies, where colds are rife, and he should form no habit that would tend to arouse the latent susceptibility of neurotic spasm.

Section 2: *Curative or ameliorative.*—In the earlier stages of the disease itself, we adopt the usual guide in the treatment of all diseases. We first of all endeavour to put the patient under the best hygienic conditions possible for general health and suitable for the individual and the disease. By this means we increase his natural powers of resistance to infection; we encourage to a maximum the capacity of the tissues to regain their normal status, and we lessen the unstable irritability of the nervous system, which is such a potent factor in the asthma element. The details vary in each case and so only the outlines or principles can be given; during exacerbations rest in bed in a warm equable temperature of 60° F., a light diet to meet any disturbance of the digestive system and freedom from worry or other disturbance of the nervous system are demanded, and during convalescence a residence in the South of England, as at Bournemouth, Sidmouth or Torquay, in the Channel Islands, or further afield, Madeira or the Canaries, or the corresponding artificial climate at home. The patient of means may anticipate his winter attacks, by seeking refuge in any of these places, either in October, if he be prepared

to spend the whole winter abroad, or in January if the former be impossible; January, February and March are the three most trying months. The second division of treatment is devoted to the specific treatment of the disease in question. In chlorosis we put the patient under the best hygienic conditions, and we give iron in large doses; in tuberculosis we also put the patient under the best hygienic conditions, and we give tuberculin or such other form of specific treatment as we believe in; in syphilis we regulate the patient's life and give salvarsan and mercury. In the early stages of the "pulmonary triad," at the onset of an acute exacerbation we find that warm poultices applied to the chest give relief to the dyspnœa, ease the cough, and not infrequently allow several hours' restful sleep. If we continue the poultices day after day they lose their effect, they weaken the patient and render him more susceptible to the faintest chill. Accordingly, after the first night, we apply a layer of fine cotton-wool, which has been warmed before the fire, and renew it every three or four hours. It gives the patient a feeling of comfort and warmth, and avoids the risk of chill from sweating. Internally, I do not know any medicine that will cure the affection; the usual mist. pect. stim. seems at times to help the expectoration, but so does a little warm milk and Vichy water or some of the ordinary aërated waters, or a little sal volatile (twenty drops), or a teaspoonful of good whiskey or brandy in warm milk. We must be cautious with any form of alcohol, as the feeling of relief far exceeds the real benefit, and these patients are only too apt to resort to the remedy with increasing frequency. During convalescence and in the more chronic stages we pursue the same line. The question of vaccines is one of increasing importance, and it is at this stage that we get most benefit by their use. The sputum should be examined as already detailed, and if the services of a skilled bacteriologist are available, a compound vaccine should be made of the pathogenic organisms which are found. A dose beginning at about 5,000,000 of each microbe should be given, increasing each week by 5,000,000 according to the degree of reaction aud the improvement of the patient. There are several compound catarrhal vaccines on the market, and if it is impossible to obtain a reliable autogenous vaccine, it is perfectly legitimate—indeed, it is one's duty—to try the effect of these stock vaccines. Sometimes medicated inhalations, such as pine oil in hot water of temperature of 140° F., sometimes the fumes of patent slowly burning powders, sometimes the spray of patent fluids, give great relief.

In the later stages, when the right ventricle begins to fail, the patient becomes more and more of an invalid, nutrition is impaired, and lighter and more easily digested food is required. Meals should be more frequent, smaller in amount, and often some warm milk and aërated water is required at night, so that a spirit kettle and such utensils become part of the furniture of the patient's bedroom. When the right ventricle gives up and dropsy appears, the effect of digitalis in doses varying from ɱv–xv of the tincture three times a day should be tried, but we cannot hope for much effect, as the cause of the evil is ineradicable; the tissues are becoming deteriorated from want of oxygen, from disturbed and inefficient metabolism, and from inefficient action of the eliminating organs. We have reached the final stage, when not only can we not cure, but we cannot amelioriate. If some complication does not hurry the final event, one of Nature's many forms of narcosis, mal-aëration of the blood, uræmia, suffusion of the cerebral meninges, kindly and gently deadens consciousness in the last hours, while the patient is being slowly drowned in his own sputum.

October 20th, 1913.

THE NAUHEIM TREATMENT OF DISEASES OF THE HEART AND CIRCULATION. By LESLIE THORNE THORNE, M.D. Fourth edition. Crown 8vo. Pp. 104. London: Baillière, Tindall & Cox. Price 3*s*. 6*d*. net.

It is not always possible or convenient to send heart cases to Nauheim for treatment. The object of this book is to give explicit directions how best to carry out a similar routine at home. There can be little doubt that much misconception exists as to what really constitutes a course of Nauheim treatment. This work makes it quite clear, not only in the text, but by means of excellent photographs, which illustrate each separate position in the administration of the exercises. Dr. Thorne has given the profession a useful book.

BRACHIAL PRESSURE NEURITIS DUE TO A NORMAL FIRST THORACIC RIB: ITS DIAGNOSIS AND TREATMENT BY EXCISION OF RIB.*

By JOHN MORLEY, Ch.M., F.R.C.S.,
Hon. Surgeon, Ancoats Hospital, Manchester; Lecturer in Clinical Anatomy, Manchester University.

GENTLEMEN,—This patient is a nulliparous married woman, æt. 42 years. When I first saw her in the out-patient department a few weeks ago she complained of old-standing pain in the right arm, and gave the following history: Pain in the arm began to trouble her about twelve years ago, when she was thirty years of age. The trouble came on gradually without any injury or other obvious cause to which she could attribute it, and steadily grew more severe until, during the last few years, it has made her more or less an invalid. Let me ask you particularly to notice the character of the pain that has given her so much trouble. She described it as a more or less constant heavy dragging pain running from the supra-clavicular fossa and right shoulder down the inner side of the arm and forearm into the ring and little fingers. It was varied at times by a sharp shooting pain that had usually the same distribution, but sometimes affected the outer fingers as well. She had also been subject at times to painful cramps affecting the small muscles of the hand, particularly those of the hypothenar eminence. These cramps were generally associated with a feeling of "pins and needles" and deadness in the ring and little fingers. When we investigated the factors that influenced the severity of the pain, she told us that she is naturally right-handed, and that the pain is always made worse by using her right arm, but particularly by carrying a heavy weight with the arm hanging by her side. From the first she found that relief could be obtained when she raised the shoulder by sitting in an arm-chair or placing the arm in a sling. Of late years she has been compelled to keep the arm in a sling whenever she walks about, and at night she has found it necessary always to sleep on the affected side. For a long time she has been under treatment for "rheumatism" and "neuritis." Counter-irritants, massage, electricity and radiant heat, a course of baths at Buxton and various drugs internally have all had their turn. Nothing, however, but resting the arm in a sling has given her material relief. Her general health has been fairly good, except for an occasional attack of influenza.

The history then was, briefly, pain down the inner side of the arm, aggravated by depressing, and invariably relieved by elevating the shoulder.

On examination, we found that there was no perceptible anæsthesia, either to a pin-prick or the light touch of a wisp of cotton-wool. The peripheral nerves were not tender on palpation of the arm, nor was there any palpable mass in the axilla, but on deep pressure above the middle of the right clavicle she experienced the pain shooting down the inner side of the arm, of which she had told us, and the same pain was elicited by dragging on the arm so as to depress the shoulder. There was no obvious muscular wasting, but the right little finger was held in a position of slight abduction, and there was pronounced weakness of adduction of all the fingers on the right side as compared with the left. The grip of the right hand was weaker than the left. The radial pulses were equal, and there was no disparity in the colour of the hands; nor did the patient complain of any vaso-motor troubles such as attacks of pallor and coldness of the hands.

Now consider the *diagnosis* of this case. The distribution of the pain down the inner side of the arm in the areas supplied by the ulnar, internal cutaneous and lesser internal cutaneous nerves suggested a lesion of the lowest trunk of the brachial plexus, formed by the eighth cervical and first thoracic nerves (see Fig.), and the weakness of the interossei strengthened this suspicion. The influence of depression of the shoulder and of palpation in the supra-clavicular fossa in aggravating the pain was a strong reason for attributing the trouble to a cervical rib. In that not uncommon condition, as you are aware, the inner border of the accessory rib, formed in connection with the seventh cervical vertebra, is apt to cause pressure on the lowest trunk of the plexus as it passes over the bone, and this pressure may be manifested by sensory, motor or vaso-motor disorders in the arm. In this case vaso-motor symptoms were absent and motor symptoms slight, but it certainly seemed to be a typical instance of the neuralgic type of manifesta-

* Post-graduate Lecture delivered at Ancoats Hospital.

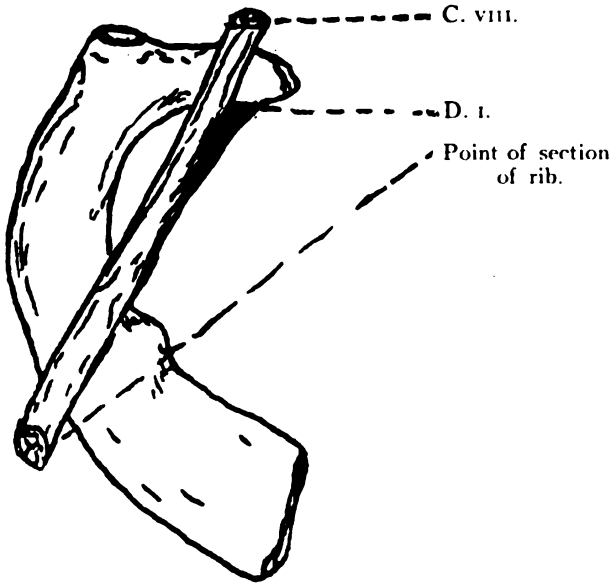

Diagram showing normal relation of lowest trunk of brachial plexus to first rib. (*After* Wood Jones.)

tions of a cervical rib. The patient was sent to our honorary radiographer, Dr. Bythell, and I was somewhat surprised to learn that the skiagram showed no cervical rib at all. The negative result of the radiographic examination could not, however, refute the reasoning by which we decided that the patient's troubles were due to mechanical pressure on the lowest trunk of the brachial plexus. Thorburn some time ago described a case (1) in which, when operating for a supposed cervical rib, he removed part of a normal first thoracic rib with perfectly satisfactory results. A case was also described by Thomas Murphy (2) in 1910, in which symptoms of pressure neuritis occurred in the absence of a cervical rib, and excision of a normal first rib was undertaken with complete relief to the patient.

Further, it has been pointed out by recent writers on the subject that a certain proportion of supposed cervical ribs prove, when the vertebræ are carefully enumerated from the atlas downwards by the help of the X rays, to be in reality rudimentary first thoracic ribs; and if a *rudimentary* first rib can occasionally cause pressure symptoms, it seemed reasonable to suppose that, given a similar disposition of the plexus and shoulder girdle, a fully developed rib might do so still more readily.

Influenced by these considerations I advised an operation, to which the patient readily gave her consent. Accordingly, under ether anæsthesia, an L-shaped incision was made down the anterior border of the trapezius to the clavicle, and forward above that bone to its mid point. A flap of skin and fascia was raised, and first the trapezius and sterno-mastoid and then the posterior belly of the omohyoid and the outer border of the anterior scalene muscle were defined. In exposing the middle and posterior scalenes the transversalis colli vessels, which were in the way, were ligatured and divided. By separating the posterior and middle scalenes the outer border of the rib was now exposed, and working as far as possible subperiosteally, first its upper surface, which is crossed by the lowest cord of the plexus and by the subclavian artery, was cleared, and then its lower surface, which rests on the pleura. The rib was normal in size and shape, with a slight *sulcus subclaveæ*. The tubercle of the rib was now disarticulated from its union with the transverse process of the vertebra and the neck cleared. The rib was now lying free and bare from its neck as far forward as the scalene tubercle. It was next divided transversely across the scalene tubercle, seized with lion forceps and twisted out, in which process it was fractured across the neck. The rest

of the neck was removed piecemeal, and part of the head, which could cause no possible trouble, was left *in situ*. You will see that by this procedure all that part of the rib in contact with the brachial plexus was removed, so that the nerves sank down and rested on the pleura. The wound was closed without drainage, and healing occurred by first intention.

The result of the operation has been on the whole very satisfactory. The constant dragging pain that she complained of has not been felt since, and she has quite lost the shooting pains down the arm and cramps in the hand that formerly gave her so much trouble. She can now walk with the arm swinging freely by her side, and there is no perceptible weakness in the grip nor in the intrinsic muscles of the right hand. She has still at times a feeling of pins and needles in the fingers, but that is growing less noticeable. She complains, however, of some tenderness over the point of the shoulder that has troubled her since the operation, and this is due probably to an injury to the acromial sensory branches from the third and fourth cervical nerves, inflicted when the incision in the posterior triangle was made.

Now how are we to explain the pressure symptoms in these cases where no supernumerary rib is present? What anatomical anomaly is the essential cause of such a malady assailing a woman in the prime of life? Wood Jones (3) has suggested that the occurrence of rudimentary first thoracic ribs simulating the commoner seventh cervical ribs is due to a post-fixation of the brachial plexus, whereby the second thoracic nerve contributes to the lowest trunk, and considers that the pressure of the post-fixed trunk in early life prevents the normal development of the rib. He believes, too, that seventh cervical ribs are associated with a pre-fixation or shifting in a cephalic direction of the plexus, so that the usual contribution to the plexus from the first thoracic nerve is reduced or absent. To account for cases such as this in which pressure symptoms are produced by a normal first rib, he makes the suggestion that there is a slight degree of caudal shifting or post-fixation of the plexus, so that the first thoracic nerve gives an unusually large contribution to the lowest trunk; and this trunk, though not exerting such pressure on the rib as to stunt it during the plastic stage of growth, may yet suffer pressure from it in later life. I was not able to test this theory by examining the size of the contribution from the first thoracic nerve in the present case, because free exposure and dissection of the plexus was unnecessary and would seriously have complicated a somewhat dangerous operation. But while we may admit on general grounds that the more the plexus is displaced in a caudal direction, the greater will be the tendency to pressure of the lowest trunk on the first rib, over which it must arch, yet it is by no means established that this anomaly of post-fixation is an invariable factor. Nor would the theory by itself account for the onset of symptoms in early adult life, and their greater frequency in women.

Wingate Todd (4) lays emphasis rather on the descent of the shoulder that normally takes place from infancy to adult age. He points out that the further the shoulder drops, relatively to the thoracic operculum, the greater will be the tendency for the lowest nerve-trunk to be stretched over the highest rib; and by careful measurements he has shown that in women, whose muscular development is, of course, slighter, the shoulder drops a good deal lower at adolescence than it does in men. In the normal individual this tendency to nerve pressure from dropping of the shoulder is counteracted and obviated by a descent of the anterior extremity of the first rib, but these two factors work independently. If an individual is unfortunate enough to combine a relatively great descent of the shoulder with a relatively slight descent of the rib, the nerve-trunk is in danger. Here, then, is our explanation of the onset of symptoms in early adult life and in females rather than males.

It is possible that when we have learned to recognise at an early stage the significance of symptoms such as I have described in this case, we may succeed in counteracting this tendency to excessive dropping of the shoulder by developing the trapezii and levators of the scapulæ by systematic exercises, and so obviate an operation. But in the case of this woman gymnastic exercises would have been intolerable, and operation gave the only reasonable prospect of relief. The case is of interest as showing you that pressure neuritis in the arm may occur in the absence of any super numerary cervical rib, and that removal of an offending rib, though not an operation to be lightly undertaken, is capable of affording relief from a most painful and disabling malady.

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(1) Thorburn, W. — 'Dreschfeld Memorial Volume' (University of Manchester Publications, No. xxxv), 1908, pp. 85–111.

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(3) Jones, F. Wood.—'Proc. Roy. Soc. Med.,' vi, 1913, pp. 95–113.

(4) Todd, T. Wingate.—"The Descent of the Shoulder after Birth," 'Anatomischen Anzeiger,' Band xli, 1912, p. 385.

October 20th, 1913.

Manual of Surgical Treatment. By Sir W. Watson Cheyne, Bart., C.B., Hon. Surgeon in Ordinary to H.M. The King, and F. F. Burghard, M.S.Lond., F.R.C.S., Surgeon to King's College Hospital. New Edition. Revised. Pp. 620. London: Longmans, Green & Co. 21*s*. net.

This, the fourth volume, is re-written with the assistance of J. P. Legg, M.S., Assistant-Surgeon, King's College Hospital, and Arthur E. Edmunds, M.S., Surgeon, Great Northern Central Hospital, and deals with the treatment of the surgical affections of the jaws, tongue and alimentary canal. Its importance to the general practitioner is great and obvious, since subjects like hæmorrhoids, hernia, intestinal obstruction, tonsils, adenoids, appendicitis, etc., are reviewed. For actinomycosis iodide of potassium in large doses is relied on, with subsequent exposure of the affected area to the action of X rays. We may mention that good results have lately followed the use of a vaccine. There is an excellent and exhaustive description of epithelioma of the tongue and the removal of the disease. Adenoids, it appears, attack the Jews in greater proportion. This applies to other maladies. In Chapter XXVI, devoted to appendicitis, much sensible matter will be found, which the reader would do well to mark, learn, and inwardly digest. The advice given should efficiently guide him through the difficulties which beset the treatment of this disease. There is food for reflection also in Chapter XXVII, "The Acute Abdomen and its Treatment." We repeat, this is a volume for the general practitioner, one he should possess and peruse.

A Text-book on Gonorrhœa and its Complications. By Dr. Georges Luys. Translated and edited by Arthur Foerster, M.R.C.S., L.R.C.P., late Resident Medical Officer, London Lock Hospital. Royal 8vo. Pp. 384. London: Baillière, Tindall & Cox. 15*s*. net.

"Gonorrhœa is one of the scourges of humanity, which so far has had but slight attention from the general public."

"Gonorrhœa is no benign disease which calls for jocular treatment. It is a serious illness which may terminate fatally."

"Gonorrhœa deserves the fullest attention of medical men and public bodies, owing to the slow but sure havoc which it causes in the individual."

These extracts from the preface indicate Dr. Luys' opinion of the disease and its results. To stimulate, aid and guide those responsible for the health of the race in a campaign against the plague is the object of the book, and certainly the abolition of the evil calls for militant methods.

On p. 15 it is stated that no less than 800 out of 1000 husbands in New York suffer from this malady, and that 90 per cent. of them have not been cured. There can be little doubt that gonorrhœa has not been, and is not even now, treated with the care it deserves. In many cases the treatment is begun, continued, and sometimes ended in the hands of the local chemist, there being a distinct aversion to consulting a medical man, especially the family doctor, until some of its complications and sequelæ manifest themselves and make further concealment an impossibility.

One chapter is assigned to the disease as it occurs in women and children. The two last chapters describe in detail the treatment of the acute and chronic conditions with the methods which have stood the tests, and which if properly and continuously applied lead to a cure, for no case is incurable in this writer's opinion. The complications of gonorrhœa are fully discussed along with the appropriate treatment.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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INGUINAL HERNIA IN THE CHILD.*

By H. S. CLOGG, M.S.Lond., F.R.C.S.Eng., Surgeon, with care of Out-patients, Charing Cross Hospital; Senior Surgeon to the Evelina Hospital.

INGUINAL hernia in the child is, I believe, always dependent upon a developmental error, the sac of the hernia being the processus vaginalis, in whole or in part. The hernia descends the whole length of the inguinal canal, and is, therefore, of the oblique or *indirect* variety. I have never seen a case, nor have I seen the report of a hernia in childhood which came directly through the posterior wall of the canal, a type which in the adult is known as a *direct* hernia, and is an acquired defect.

The processus vaginalis is the patent protrusion of peritoneum which precedes the descent of the testicle from the inguinal region into the external genitals. The testicle in its descent invaginates into the process posteriorly. The processus in the male extends to the bottom of the developing scrotum, and in the female, where it is called the *canal of Nuck*, into the labium majus.

It is generally conceded that the processus vaginalis in normal development should become separated from the peritoneum at or shortly after birth. The separation takes place first immediately above the testicle (the lower portion remaining as the tunica vaginalis), and then at the site of the internal abdominal ring. The intervening portion becomes gradually obliterated, but may in part remain as a fibrous cord which can be dissected as one of the constituents of the spermatic cord. The failure of this processus to undergo normal development accounts, I believe, for all inguinal hernia in childhood, and for some forms of hydrocele of the cord.

* A lecture delivered at the Polyclinic.

Perhaps some will not agree that all inguinal herniæ in children are dependent upon the remains of the processus vaginalis, but regard some herniæ as acquired. This supposition implies some weakness of the abdominal muscles and some increased intra-abdominal pressure, which two factors acting together will cause a gradual protrusion of the peritoneum through the weak spot of the abdominal wall, viz. the internal abdominal ring, where the cord passes from the abdominal cavity into the scrotum. Inguinal hernia is found both in the healthy and the weak child; it is found alike in those who have, or have had, abdominal distension from gastro-intestinal disorders, and in those who have not, in those with phimosis or other conditions which cause straining efforts with increased intra-abdominal pressure, and in those not so affected. It may be seen for the first time during the course of some wasting illness, but this does not prove that the sac is acquired. Naturally if the sac were preformed the hernia would descend during a wasting illness, when the muscles are weakened. At operation upon these herniæ the muscles will nearly always be found to be well developed, except in the large herniæ, where constant pressure will have stretched and thinned the structures to some extent; this is a secondary effect. A hernia when first noticed is often of large size, and this is particularly the case in wasted babies. Obviously the sac in such was preformed, for if acquired, it would gradually form and progressively extend in size. In many cases a hernia will only descend at intervals, whereas if it were acquired it would constantly be present. The age at which the hernia appears is no criterion of the origin of the sac. A congenital sac may be unoccupied if the neck is comparatively narrow, and well guarded by the internal oblique muscle. In children the neck of the sac is frequently quite narrow, and the internal oblique muscle well developed, and hence the hernia will only descend at intervals when the internal oblique muscle is taken off its guard. Hence it is that when a child is brought with the history of a swelling which has disappeared, it is frequently impossible to get the hernia to descend and to demonstrate its presence. It is quite common, even in adults, for a hernia to descend for the first time, so history relates, into an unobliterated processus vaginalis.

All these facts support the contention that the hernia is congenital, but I think the most convincing are those obtained from an examination of the interior of the sac. In the great majority of cases there will be seen in the interior of the sac some irregularities of the surface often amounting to constrictions, or imperfect septa, which can be interpreted only as developmental processes, and can therefore only occur in a congenital sac.

Granted that the sac is of congenital origin, in the male two types may be recognised: (1) The sac is the complete processus vaginalis—the *complete* sac—and extends to the bottom of the scrotum, having the testicle projecting into it posteriorly, as it does into the tunica vaginalis. The contents of such a sac will more or less completely surround the testicle and obscure it; upon this fact the clinical diagnosis of this type of hernia depends. (2) The lower part of the processus vaginalis has become separated, the upper part only remaining in communication with the peritoneum—the *incomplete* sac. The size of the incomplete sac will vary considerably, depending upon the site at which it has been naturally obliterated, and also upon the increase in size which has been allowed to take place for want of treatment. It will often extend to the tunica vaginalis, and be intimately adherent to it, showing that obliteration has taken place only immediately above the testicle. On the other hand, it may be merely a small sac in the inguinal canal having no connection whatever with the tunica vaginalis. All intermediate stages are seen. The incomplete sac when allowed to increase in size may pass down in front of the tunica vaginalis, or in some cases behind it, and in the latter before exposing the sac the tunica may be opened. In other cases it may invaginate itself into a capacious tunica vaginalis, and form what is usually called an *encysted* hernia. These relationships to the tunica should be borne in mind, otherwise confusion may arise at operation.

It is commonly stated that a hernial sac in infancy and childhood is the complete type. This is entirely wrong. I should estimate, from an experience of some 400 operations, that a complete sac is present in something less than 10 per cent. of cases. Herniæ associated with imperfect descent of the testicle are excluded; in such herniæ there is always a sac, and it is complete.

In the female the sac extends almost invariably into the external genitals, the canal of Nuck

remaining in its entirety. In this sac, as in the male, irregularities, constrictions or imperfect septa are seen, and must be explained on developmental grounds.

An extension of the sac between the peritoneum and muscles, between the oblique muscles, or between the muscles and subcutaneous tissues, *i.e.* the so-called interstitial sac, I have not seen in children save in association with imperfect descent of the testicle. In the latter abnormality it is common to find a sac superficial to the muscles—the extra-parietal variety of the interstitial sac lying altogether superficial to the inguinal canal—in a position commonly assumed by an imperfectly descended testicle. If the scrotum be imperfectly developed this interstitial sac will represent the whole of the processus vaginalis, or the latter may occupy a developed scrotum, the interstitial sac being an offshoot from it as it were.

It should be mentioned that an interstitial sac has been observed in cases where the testicle is completely descended, but is extremely rare; it has been seen in the female child.

Diagnosis.

This may be considered from two standpoints:

(1) *The history of a swelling which has been seen and has disappeared, and cannot be seen when the child is brought for examination.*—I have already stated that it frequently happens that the internal abdominal ring is so well guarded by the internal oblique muscle that the hernia descends only at intervals, and in spite of energetic exercise cannot be made to appear when required. The diagnosis of a hernia upon history alone should not be made too hastily. Glands come suddenly, or are noticed suddenly, and disappear; hydroceles sometimes arise acutely in children and vanish; parents may mistake the excessive pubic fat for some abnormal swelling; the testicle may slip up the canal and be mistaken for a hernia; it should be mentioned that in the latter case there is often a patent processus vaginalis.

The size of the external abdominal ring has nothing whatever to do with the diagnosis of the presence of a hernia, for the hernia depends upon development error, and not upon muscular weakness. In large herniæ the ring will naturally be enlarged, but in such cases the swelling easily protrudes. The spermatic cord may feel thickened due to the presence of a sac, but no reliance can be placed upon this for diagnostic purposes, for the sac-wall may be too thin to cause any perceptible thickening of the cord, and even if a thickness be felt it may be due to a capacious *tunica vaginalis* which extends up the cord. The diagnosis of a hernia should never be made upon history alone.

(2) *A swelling is present.*—This has such definite characteristics that there should never be any doubt about the diagnosis, but occasionally there seems to be. It is easy to determine that the swelling comes from the abdomen by being unable to detect the cord free above the swelling.

It would seem that a hydrocele is sometimes confounded with a hernia. But in all cases it is possible to differentiate that the swelling has not descended from the abdomen by being able to palpate the cord distinctly above the swelling.

Treatment.

This may be considered under two headings: (1) the *rôle* of the truss, and (2) operative.

(1) *Truss treatment and its value.*—I have already mentioned that we are taught that the processus vaginalis in normal development should be obliterated at or shortly after birth. Peritoneal development goes on after birth, and I shall have occasion to refer later to some of the changes which occur in the peritoneum after birth. Briefly here I may state that during development certain layers of peritoneum may fuse together and that layers of peritoneum may disappear, and that such changes in the peritoneum occur during the first few months of life. The processus vaginalis, being originally peritoneum, may conceivably therefore undergo complete development after birth, *i. e.* it may become naturally obliterated. Up to what age such obliterative changes may be anticipated is merely a matter of conjecture. Obliterative changes could obviously not take place if the hernia were allowed to descend. The only possible value of a truss, therefore, is in restraining the descent of the hernia and thus allowing Nature to proceed with normal development. A truss may act, therefore, indirectly in the cure of a hernia. As a direct cure it has no value. Its pressure cannot possibly have any influence in forming acquired adhesions in the sac, which may eventually lead to the cavity disappearing, or even in obliterating the patency

of the neck of the sac and excluding it from communication with the peritoneal cavity.

During the first few months of life I advise a truss, solely with the object of restraining the hernia, and in order that this may be effective it must be worn constantly. I advise this treatment, except in special circumstances, up to about the fourth or sixth month. I confess to having no proof that the natural cure may not take place at a later age than this, but if a hernia be seen at this age I deem it exceedingly improbable that a natural cure will result, and in the absence of any contra-indications advise operation.

Operative treatment.—If a hernia is seen at or after about the fourth or sixth month of life and the child is healthy I advise operation. The operation at this age is quite well borne, the anæsthetic is not more risky than it is for any other operation of convenience, *e. g.* circumcision, and the wound heals perfectly. In special circumstances, *e. g.* a large hernia which cannot be controlled by a truss or a hernia which is frequently coming down and causing difficulty in reduction, operation will have to be undertaken at an earlier age. If operation means taking the child from the breast, as it does in hospital cases, it should be deferred unless there is any special indication for early operation.

The operation.—The essential factor in the operation is the occlusion of the neck of the sac, which must be done in such a way that the peritoneum in the region of the internal abdominal ring is not left slack with a tendency to project into the inguinal canal, but is drawn taut and fixed in that position. My method of operating for the reducible hernia is briefly as follows: The external oblique is incised the whole length of the canal to thoroughly expose the neck of the sac. I consider free exposure of the neck of the sac essential; otherwise it cannot be properly dealt with. The sac should always be opened. The incomplete sac I remove entirely. I separate the complete sac from the cord a little above the testicle, ligature the lower part and leave it as the tunica vaginalis, and remove the upper part. The only difficulty in the operation is the separation of the sac. The latter in infants and children is extremely delicate, and tears very readily; it has several coverings derived from the tissues of the abdominal wall. If separation is commenced in the wrong layer, *i. e.* if by mistake it is attempted to separate the coverings with the sac, considerable difficulty will be experienced. But if the edge of the opened sac be identified and separation of this be commenced it can be very easily and readily accomplished by gently brushing the coverings from it with gauze. Having isolated the sac as high as the neck I treat it by one of two methods. In the majority of cases I apply forceps to it, torsion, and ligate it. The stump of the sac springs into the abdominal cavity and the slack peritoneum at the site of the internal abdominal ring is braced up tightly. In some cases, for instances in older children, in some cæcal and bladder herniæ and in some of the ovary and tube I invaginate the sac according to Kocher's method. But I am sure that the torsion and ligature is much easier, and, save in exceptional circumstances, quite as effective as is Kocher's method. I usually place one suture to approximate the internal oblique and torn cremaster muscles to Poupart's ligament over the cord at the site of the internal abdominal ring. The external oblique aponeurosis is sutured, also the fat and the skin. As a dressing I always apply sealed collodion, and have not seen any disadvantages from its use.

It is quite common to find a little clear fluid in these hernial sacs, which seems to have no importance. On two or three occasions in addition to fluid I have seen the sac studded with miliary tubercles. Such cases were evidently instances of latent and overlooked peritoneal tuberculosis.

The vast majority of herniæ in children are reducible, but occasionally the contents may be in whole or in part irreducible. The causes of irreducibility are adherent omentum, some cæcal herniæ, adherent appendix, a variety of hernia of the iliopelvic colon, extra-saccular hernia of the bladder, some herniæ of the ovary, and strangulation of the contents.

Hernia of the Cæcum and Appendix.

The cæcum is frequently herniated in children. I have often seen it prolapse during operation, and have frequently when a piece of gut shows itself at the neck of the sac drawn it down, out of curiosity, and found it to be the cæcum. The cæcum has been seen in a left-sided hernia, but I have never had an example of this rarity. The frequency of cæcal hernia in childhood is readily explained. The cæcum is formed as an outgrowth from the large gut, and is covered entirely with peritoneum,

but has no mesentery. The ascending colon has originally a mesentery which is attached to the posterior parietal peritoneum of the right side of the abdomen. Later this mesentery fuses with that parietal peritoneum upon which it lies, and the fused layers disappear, leaving the ascending colon denuded of peritoneum posteriorly. This is usually regarded as the normal development of the peritoneum. Some time ago I examined a considerable number of bodies of children of all ages, and found that in infancy the ascending colon nearly always retained the original mesentery, whereas in older children the presence of a distinct mesentery was much less frequently observed. This is borne out by hernial statistics, for it is in the infant that I have most often seen the cæcum in the hernial sac. The cæcum is situated in close apposition to the internal abdominal ring and its descent is readily permitted when the ascending colon is mobile as it is when provided with a mesentery.

When the cæcum prolapses into the sac in this manner it may be called a simple hernia to distinguish it from a variety to be mentioned later. The relationship of the cæcum to the sac is similar to that of the small intestine. The appendix may be a hernial content with the cæcum, or the appendix may alone descend into the sac. Hence arises the possibility of appendicitis in the hernial sac.

When operating upon right-sided herniæ, if I see the cæcum, I drawn it down with a view to exposing the appendix. If the latter can be drawn down sufficiently to expose its cæcal attachment I remove it, as one does during the course of an abdominal operation when accessible.

The second type of cæcal hernia is rare in childhood; it is uncommon in adults; it is always on the right side. In this type the cæcum and perhaps the ascending colon in part prolapse into the sac in such a manner that the mesentery of the colon is attached to the posterior wall of the sac. It would seem as if the cæcum, the colon and its mesentery with the parietal peritoneum had been displaced from its normal position into the inguinal canal and scrotum. The sac may contain, in addition to the cæcum and perhaps the colon, some small intestine. If the latter the contents are in part reducible, but if the cæcum and colon are the sole content they cannot be reduced on account of their attachment to the posterior wall of the sac. An irreducible hernia on the right side containing bowel and not strangulated should at once excite suspicion that it is this type of cæcal hernia.

In the adult, where this type of hernia is relatively more common, it is probably explained by a prolapse of viscera, the colon retaining its primitive mesentery (or according to some gaining an acquired mesentery by the constant drag upon its peritoneal attachments). In such the cæcum, which would lie close to the internal abdominal ring, descends into a hernial sac, and as the latter enlarges it does so at the expense of the parietal peritoneum in the neighbourhood, thus drawing the colon and its mesentery into the sac. In the child these herniæ do not reach the size to which they attain in the adult, and visceral prolapse does not occur, or certainly not to the same extent. They must be explained upon developmental grounds. According to Lockwood there may be a fœtal fold passing from the mesorchium to the cæcum, lower ileum and mesentery. If this fold persists the testicle in its descent would drag upon the cæcum and the peritoneum of the posterior abdominal wall and draw these structures into the inguinal canal. Some state that adhesions may form between the posterior surface of the cæcum and the peritoneum covering the undescended testicle, and the latter in its descent draws the cæcum into the canal.

A further degree of this type of hernia occurs in adults, but I have never met with it in childhood. The mesentery of the colon has disappeared, and the colon lies with its posterior surface denuded of peritoneum directly upon the posterior wall of the parietal peritoneum. In the adult as the sac enlarges it may drag the cæcum and colon down into it in such a manner that the colon lies directly upon the posterior surface of the sac. This is an extra-saccular hernia—that is, the cæcum and colon are in part outside the sac. Theoretically this would be possible in childhood if peritoneal fusion had occurred, and would be explained either by the drag of the band described by Lockwood or by adhesions between the cæcum and testicle. But it must be extremely rare in childhood, if it ever occurs, for the colon in early life nearly always has its mesentery, and these herniæ in children do not progress to the large size they attain in adult life.

The treatment of an irreducible cæcal hernia varies with the size of the hernia. In small herniæ in children the sac may be separated together with the cæcum, the latter returned into the abdomen

and the sac invaginated between the peritoneum and muscles and there fixed (Kocher's method of treating hernial sacs). In larger herniæ a complicated modification of technique is necessary which it is not necessary to describe here.

Appendicitis in a Hernial Sac.

An analysis of this condition shows that there are two ages at which it appears to be most frequently seen. The one is in later life, and the other in childhood. Approximately about 25 per cent. of the cases have been recorded in childhood. This is explained by the frequency of the appendix in the hernial sac in childhood, and the very great frequency of inguinal hernia in children. It would seem that an appendix in a hernial sac would be peculiarly liable to be kinked, its lumen obstructed, and vascular supply interfered with, and therefore rendered prone to infection. The condition has been observed in quite young infants of a few weeks or months old, but is relatively commoner in older children.

Clinically I have seen appendicitis in the sac in two ways:

(1) *A chronically inflamed appendix adherent to the sac wall.*—The appendix in my cases has formed the sole content of the sac, but of course the cæcum, or perhaps small intestine, may also be present. If these are present they may be reducible. The adherent appendix is felt as a thickened strand passing from the scrotum into the inguinal canal. There may or may not have been any complaint of pain in the hernia. In one of my cases the boy was wearing a truss, and said that he was unable to wear it on account of the pain. What relation the truss bore to the diseased appendix I cannot say.

The treatment of this condition is simple. The appendix is isolated and removed. It may not be possible after freeing the appendix to draw the cæcum sufficiently down into the sac to expose the attachment of the appendix, and in such a case a muscle-splitting incision immediately above Poupart's ligament should be made, and through it the appendix removed.

(2) *A definite abscess is present.*—This is more common in my experience than the former variety.

The diagnosis of the condition is not always easy. The onset of the illness is acute, and if the child is old enough to describe his symptoms, they will be acute abdominal pain, centrally situated, with or without pain in the hernia in the early stages. If looked for, a swelling will be present early in the inguinal region, or scrotum, or in both: or if a hernia previously existed there will be a sudden increase in its size. The temperature is elevated. The onset of an acute appendicitis in a hernial sac may be less acute than when the lesion is within the abdomen. I have had one case where an abdominal appendicular abscess tracked down a patent processus vaginalis, forming also an abscess in the hernial sac. These cases are usually mistaken for strangulated herniæ. At first the similarity is great. The acute onset, the sudden swelling and peritoneal symptoms are common to both. In appendicitis we have to deal with a primary acute inflammatory lesion, whereas in the strangulation a primary obstructive lesion is present. In the inflammatory lesion there will be early œdema of the tissues, an ill-definition of the swelling, which is very tender, and elevation of temperature. In the obstructive lesion the swelling is well defined, the coverings in the early stage do not show œdema, and there is not such acute tenderness in the swelling, and the temperature is subnormal. Shock is more pronounced in the strangulation.

A further condition which may be mistaken for appendicitis is torsion of the spermatic cord. This occurs occasionally in infants and very rarely in the older child; relatively it affects an imperfectly descended testicle rather than one of full descent. The onset is acute, but abdominal symptoms are absent. If the testicle be imperfectly descended and lies in the tissue superficial to the inguinal canal, the swelling will be rounded and at first well-defined, and it may be possible to feel the external abdominal ring, showing the canal is empty. If the testicle is in the scrotum it can be ascertained that the inguinal canal is empty. The temperature is not raised in the early stages.

The Iliac and Pelvic Colons.

On the left side the pelvic colon may engage in a hernial sac as the cæcum does on the right side. On a few occasions I have seen the pelvic colon escape through the ring during some straining effort at the operation. The pelvic colon is not in close proximity to the internal ring and hence is not so frequently herniated as is the cæcum. Such a hernia is quite simple and demands no further mention.

In a second type of hernia the colon is attached by its mesentery to the posterior wall of the sac. As in the cæcal hernia, such a condition is more frequently observed in the adult, but occasionally it may be seen in the child. The reasons for its occurrence in the adult are similar to those of the cæcal hernia. In the child probably the cause is a fœtal band similar to that which may cause a cæcal hernia, the band being attached to the peritoneum and colon, so that when the testicle descends it drags the colon down with it. In my experience the hernia is extremely rare: I have met with the condition only once as far as I can remember and then only slightly marked. The size of the hernia must vary, and as on the right side in addition to the colon small intestine may descend into the sac. It may therefore be in part reducible. An incompletely reducible hernia on the left side containing bowel should excite suspicion that the pelvic colon and its mesentery in part may be the contents.

In the child merely a small portion of colon will occupy the sac, and the length of mesenteric attachment will be comparatively short, but in the adult larger herniæ of this type occur in which there is a large loop of colon and an extensive attachment of mesentery. Should the iliac colon also descend and this be denuded of peritoneum as it is in part in adult life, then the hernia may be in part extra-saccular, as the hernia of the ascending colon may be. But I do not think this condition will occur in the child, for the herniæ do not reach these large dimensions, and the iliac colon in early life usually has a free mesentery.

Bladder.

The bladder, covered with peritoneum superiorly and posteriorly and uncovered inferiorly and laterally, may occupy a hernial sac in one of two ways. The part covered with peritoneum may prolapse precisely similar to the way in which intestine does—that is, it may be a reducible content. The bladder being situated relatively higher in the infant than in later life, being more of an abdominal organ than a pelvic one, it might be thought that this form of hernia would be a relatively more common condition in the child than in the adult. It is not, however; I have not seen an example of it. In the second type of hernia the bladder forms a part of the sac-wall—an extra-saccular hernia. When operating upon herniæ in children, and separating the sac at the internal abdominal ring, on several occasions I have drawn the bladder just into sight. These are not instances of hernia of the bladder, but show how it is possible for the bladder to become herniated, which I have seen on two or three occasions. If a sac increases in size it does so at the expense of the parietal peritoneum, and if the peritoneum on the inner side is drawn down into the sac, the bladder, being intimately attached to it, may come to form part of the inner wall of the sac. The descent of the bladder is preceded by the extra-peritoneal fatty tissue. The relatively high position of the bladder explains the greater frequency of this type of hernia in the child. The herniated portion of bladder in the child is too small to be detected clinically in my experience. Should it be of large size, it might be felt as a firm thickening after the contents of the sac have been reduced as it has been in the adult. In the latter also disturbances of micturition had sometimes been recorded, and pressure upon the swelling has caused a desire to micturate. In the child none of these phenomena have been observed and the condition will never be diagnosed.

In every operation the possibility of a herniated bladder must be borne in mind. Before ligating the sac it should always be carefully inspected and palpated at the inner margin, and if unduly thickened there suspicion should be aroused. A useful guide as to the proximity of the bladder is the presence of extra-peritoneal fat at the inner aspect of the neck of the sac, for this precedes the descent of the bladder. It is by no means easy to recognise the muscular fibres of the bladder-wall, and unless great care is always exercised accidents will sometimes happen.

The treatment of this condition in the child is usually simple. The portion of bladder is usually so small that it can easily be separated from the tissues around and returned into the abdomen. The sac after isolation is invaginated. It is advisable to suture the walls of the inguinal canal as in the operation upon the adult. If the herniated bladder is of larger dimensions some complication of technique may be necessary.

Ovary and Tube.

The ovary and with it often the fimbriated end of the Fallopian tube is a frequent content of a hernial sac in the child, whereas it is a very rare

one in the adult. The younger the child the more often have I noticed the ovary as a content of the sac. I have also noticed that in children with hernial sacs, although the ovary may not have been known to enter the sac, it frequently lies in close proximity, for in separating the sac in the region of the internal abdominal ring it may be drawn down with the parietal peritoneum.

The herniated ovary may be reducible or irreducible. The mesosalpinx may be attached wholly to the peritoneum within the abdomen when the ovary is reducible. Sometimes, however, the attachment of the mesosalpinx may extend into the neck of the sac and be attached there, when the ovary may be irreducible. Occasionally the tension of the structures bounding the inguinal canal may prevent reduction.

Clinically the herniated ovary may easily be diagnosed by its small rounded or oval shape and its mobility. It is usually the sole content of the sac.

The treatment is quite simple. The sac is separated from the surrounding tissues, with the mesosalpinx, if this arise from it, and invaginated according to Kocher's method. The ovary must not be removed unless strangulated, or unless its pedicle is torsioned and it is damaged beyond recovery.

Strangulation.

It is quite common to have children brought to the hospital with the history of a sudden descent of a hernia, and in which, judging from the symptoms, the contents are lightly snared. Such herniæ in the majority of cases can be reduced by gentle taxis, often without an anæsthetic, but if there is the slightest difficulty an anæsthetic should be given, as this will relax the muscles which are usually the cause of strangulation. A small minority of such herniæ cannot be reduced, and are definitely strangulated. Relatively strangulation is most frequent in the first six months of life, and it is within the first two months that the greater number of cases are seen. Several cases have been recorded within the first few days of life. After the sixth month its frequency relatively declines, in children over two years it is uncommon, and in older children rare.

The *diagnosis* of a strangulated hernia is usually readily made. The sudden appearance in the inguinal canal and scrotum of a rounded, tense, elastic swelling, dull or resonant to percussion according to whether there is or is not fluid in the sac, associated with the familiar constitutional symptoms, is quite characteristic. Occasionally it would seem that an acute glandular swelling may be mistaken for a strangulated hernia. It may be readily differentiated from it by noting the position of the swelling, its character, the accompanying signs of inflammation and probably elevation of the temperature. The differential diagnosis from acute appendicitis in a hernial sac has already been mentioned.

A strangulated hernia has to be differentiated from torsion of the spermatic cord. In the latter the inguinal canal is empty, the swelling in the early stages is entirely different from that of a strangulated hernia, and abdominal symptoms are absent.

The *treatment* is, of course, immediate operation. Shock must be appropriately treated by subcutaneous saline injection and other remedies before, during, and, if necessary, after the operation.

In nearly all cases it is the bowel which is strangulated, and most usually the small intestine. In right-sided herniæ occasionally the cæcum may be strangulated. I have had one case where the appendix was the sole content of the sac and was strangulated. Very rarely is the omentum strangulated in children; occasionally the ovary may be strangulated.

The actual cause of the strangulation is not always the same. I have, in one or two instances, easily reduced the hernia after division of the external oblique aponeurosis, but more commonly this alone is ineffective. In other cases after this aponeurosis has been divided and the lower border of the internal oblique muscle retracted the hernia may be reduced, and I think the tightness of the internal oblique muscle is the most common agent of strangulation. Only rarely have I had to divide the neck of the sac before effecting reduction. This is important, as an intact sac is necessary for the radical cure of the hernia, and in the majority of cases the sac may be left intact. Should it have to be divided, after reduction of the contents the incision should be sutured. Rarely will the bowel be gangrenous and require removal. Should it be, the prognosis is very grave; otherwise it is very good.

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THE OPERATIVE TREATMENT OF FRACTURES.*

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I PROPOSE to consider the operative treatment of fractures from three points of view, attempting to answer the questions, Why? When? and How? or, in other words, to discuss the indications for operative treatment, the time at which to operate, and the methods to be adopted.

INDICATIONS FOR OPERATION.

In general, operation is needed where adequate replacement of fragments is not possible by other methods, unless the patient is unfit from age or disease. The question of what is meant by adequate reposition can be settled only by experience, varying with the situation and variety of the fracture and the age of the patient. We aim at restoring function to the limb as a whole, but correct anatomical replacement of fragments is a good starting-point for securing good function.

(1) Fractures of long bones not involving joints, particularly in children, do not need to be in absolute, perfect position, the ends of the bones fitting like the parts of a Chinese puzzle. Especially is this so if the defect is one of shortening, but if the defect is one of alteration of the axis of the limb, *i. e.* angular deformity, the defect is more serious, since alteration of the axis of the limb, especially the lower limb, causes alteration of the pressure in the joints above and below the site of fracture, setting up a series of minor injuries which ultimately will produce osteo-arthritic changes in the joint, especially in those patients of more mature years.

(2) In the case of fractures involving joints much greater nicety of apposition is necessary, since a very slight degree of failure in alignment of the cartilage-covered surfaces of the joint will, by its roughness, rasp away the opposing surface and quickly destroy the functions of the joint; consequently, where joints are involved operative treatment will be far more often necessary than in fractures of long bones.

(3) Where short bones are broken across, or processes of long bones broken off and separation of the fragments is maintained by traction of the attached muscles, operation will be needed; this also applies when any part broken off is short and not controllable by splints or extension, and is deflected from its alignment by muscular spasm. The olecranon and patella are the commonest examples of this class of fracture, and operation is more urgent in the case of the latter than the former, for the strength attained by good bony union is more essential in the lower than the upper limb.

(4) The last sentence applies equally to other fractures; absolute accuracy of "setting" is more important in the lower than the upper limb because the limping caused by a short leg and the secondary changes which are apt to supervene where there is greater strain, *i. e.* in the lower limb, renders incapacity much greater.

(5) Children have much greater power of accommodating themselves to alteration in length or deflection of the axis of a limb; an inch shortness of leg makes little difference to a child, and hence accurate reposition of fragments and operative treatment is less often needed in them.

(6) A transverse fracture once set has little tendency to become displaced again, but oblique fractures are very liable to a sliding of the fragments over each other, rendering operation and fixing necessary.

(7) In the forearm, when both bones are fractured and the ends all tend to come together, producing "cross-union" with loss of pronation and supination movements, as little can be done for this by splints, operation will be needed as a rule.

The above applies to simple fractures; where complications are present operative interference is more often requisite.

(8) In the case of compound fractures all will agree as to the necessity of operative interference in order to secure asepsis of the fracture, cleaning the wound, removing portions of bone and tissues not likely to survive. Where the fragments are readily reduced and will be maintained in position by splints nothing further is needed, but where difficulty in maintaining apposition of the fragments is experienced, as in oblique fractures, there should be no hesitation in securing them with

* Lecture delivered at the Polyclinic.

plate, screw or wire, since even if suppuration occur, the appliance inserted can probably be retained for some weeks, until sufficient callus has been formed to prevent the position of the fragments becoming bad, and this treatment does not increase appreciably the risks of spreading infection. With care and fortune a good many cases of this sort will heal by first intention.

(9) Comminuted fractures often need operative fixation to give any prospect of a useful limb, and these cases may tax the ingenuity of the surgeon to the utmost.

(10) Fractures combined with dislocation of one of the fragments often needs operative treatment, either because the dislocation cannot be reduced without operation, as in the case of fractured neck of the humerus with dislocation of the shoulder, or because the dislocation is likely to recur as in dislocation of the humerus associated with fracture of the great tuberosity. Where the dislocated portion can readily be controlled by manipulation and splinting, as in most cases of Pott's fracture—dislocation at the ankle-joint—operation will, of course, not be needed.

(11) Injuries of large vessels complicating fracture demand immediate operation if the signs point to rupture of the vessel, which then becomes the primary object of attack, ligature or suture being done according to circumstances. Where the vessel is merely pressed on, but not torn, operation will only be needed if manipulation fails to reduce the fracture and release the vessel.

(12) Where important nerves are involved it seems reasonable to explore without delay or waiting till the reaction of degeneration is present. It is difficult to say whether a nerve is bruised, torn, or nipped between fragments, and in the latter case, a usual condition, according to my experience, in the case of the musculo-spiral nerve (by far the most common nerve involved in fractures), much time will be saved by freeing the nerve when it has a chance of recovery without degeneration, while if left caught it will certainly degenerate, and results of secondary suture are much slower than when primary suture is done. Hence, the importance of not missing nerve injuries in case of fractures.

(13) Vicious union, *i. e.* union in such bad position, that function is seriously affected. This should be a rare indication in these days of radiograms : after setting a fracture or allowing extension to act for two or three days radiograms should be taken in two planes or stereoscopically, and if the adjustment is not satisfactory further attempts made by manipulation and extensions. If not satisfactory after a week or ten days, open operation should be undertaken unless strongly contra-indicated.

Where vicious union has occurred the function of the limb must be taken into account, and if fairly good it will often be well to leave matters alone, since the difficulties of operating in such cases are much greater than in recent fractures owing to the growth in size of the ends from exuberant callus and the shortening of muscles. In many cases where the defect is largely angular a well-planned osteotomy near the fracture will be better than an attempt to separate the bone at the site of fracture and re-unite in good position. Early operation in difficult cases will render operation for vicious union of much rarer occurrence.

(14) Where the ends of the fragments appear to be in good position and no union has taken place in several weeks, we may regard the case as one of delayed union ; and if, after a fair trial of vigorous massage, union is still wanting, it may fairly be considered one of non-union.

Two main conditions are found in these cases. In the first the matter is simple ; some adventitious matter interposes between the ends of the fragments, such as fascia or muscle, which cannot be removed short of open operation, and when removed the bones readily unite. In the second condition there is malnutrition of the ends of the fragments, whether from general disease or local defect, in some cases depending on deficient blood-supply, in others unexplained. Here the outlook is less good, for the broken ends become rarified, atrophic and pointed. If the atrophic part is cut away union may occur with shortening, but even after operation there may be still no union in such cases. A determined attempt at bone-grafting will be the only resource to provide a limb of proper length.

When to Operate.

The fresh cases fall into three groups. In the first come the urgent cases, including compound fractures and other urgent complicated fractures, such as wounds or obstruction of large blood-vessels, injuries of viscera, etc. These are as urgent as the acute abdomen, and should not be allowed to

wait more than the time necessary to arrange for operating.

In the second group are the cases where operation is inevitable but not urgent, including such conditions as fractured patella, cases involving nerves, comminuted and multiple fractures, or where, after an attempt at reduction under anæsthesia, the surgeon is confident that external manipulation is not sufficient to secure a good result.

In such cases some advocate waiting a week or ten days with the idea that there is less risk of "inflammation" following. Such ideas are hardly in accord with modern views of pathology. There is no inflammation of an infective nature in the repair of fractures and no increased risk in operating when recent, unless there is considerable bruising of the skin or the fracture is nearly compound, when there may be some chance of infective organisms entering the system through the damaged cuticular structures. The advantages of early operation are that less matting of tissues is present, no callus is present, and effusion of blood is less, all of which make for an easier operation, and the easier the operation the better prospect of a good result. Where much force and time are expended there is considerably more risk of some error in aseptic technique creeping into an operation. There seems, then, no necessity for delaying operation after two days in otherwise straightforward cases.

In the third group fall a large number of cases where there seems a fair chance of attaining good position by manipulation, extension, splints, etc., but in these cases it will generally be pretty obvious in a week or ten days whether or not such treatment will suffice, and if the decision is that such methods are not enough, operation is conducted without delay.

By treating cases in this manner badly united fractures should become greater rarities than they are at present.

Causes of Difficulty in Obtaining Apposition of Fragments.

(*a*) Spasm of muscles pulling the fragments apart as in fractures of the patella or olecranon.

(*b*) Spasm causing overlapping, especially in oblique fractures of long bones, *e.g.* of the femur or tibia (transverse fractures once reduced will probably remain *in sitû* with the aid of splints, etc.).

(*c*) Spasm causing angular deformity, as in the upper end of the femur and lower end of the humerus.

(*d*) Spasm causing rôtary deformity, as at the upper third of the radius.

The group (*a*) practically always demands operation; (*b*), (*c*), (*d*) may often be overcome by extension and splints.

(*e*) Entangling of the ends of the fragments in muscle or fascia: where this occurs operation will generally be needed.

Operative Methods.

In the first place the most rigid asepsis is necessary; a fracture must be treated with even more respect than the inside of the abdomen, since it is easier to infect intermuscular planes, periosteum, bone and medulla than it is to introduce sepsis into the abdomen. Handling the fragments should be avoided as far as possible even with the gloved hand; not that gloves are otherwise than sterile, but in these operations there is great risk of puncturing the glove with spicules of bone and breaking the aseptic conditions to some degree.

Reduction of Fragments.

A free incision is necessary so that the fragments can readily be brought out of the wound; in some cases reduction is easy, *e.g.* where short bones are broken across or processes separated. Where the end of bone is broken off transversely or an epiphysis separated, a thin lever introduced between the fragments will enable the surgeon to lever them into place.

Where a long bone is fractured transversely the readiest plan of reduction is to bring both fragments out of the wound, place them together at an angle in position, and force the angle thus formed to become a straight line by steady pressure on the apex of the angle: great leverage is obtained in this way.

Where the fracture is oblique this plan is not so satisfactory and traction at the end of the limb will be needed as well as levering the fragments into position. Nice adjustment so that the proper serrations of the fragments exactly fit is sometimes a matter of time and patience.

When in position the fragments may be kept

from slipping by use of large round-ended retractors. Special forceps are sometimes useful, but often get in the way of further operations.

Fixation of Fragments.

It is well to have several available methods, since it often happens that one of these will be found far the most suitable in any given case.

(1) Simple reposition is sufficient in some transverse fractures, especially in separations of epiphyses, where the cuff of periosteum displaced with the epiphysis will usually keep the latter in quite good apposition, assisted by suitable position of the limb, as acute flexion of the knee or elbow. Where the stability is in question some additional means should be adopted.

(2) The wire is the classic implement for retaining fractures in position, and still holds its ground in dealing with short bones such as jaw or patella, and may even save the situation in operations on long bones where the more modern plate leads to difficulties. The wire may be inserted through holes bored in the fragments and twisted sufficiently tight. This is most useful for short irregular bones; for long bones if used in this manner it secures apposition but does not prevent angular deformity at all well. In dealing with long bones the wire is of most use in oblique fractures, where it can be applied circumferentially, as in splicing a fishing-rod. Two encircling wires will, as a rule, suffice to secure apposition and rigidity in such a case. It may be readily applied where a plate would not fit well, from the direction of fracture, and gives excellent results; it may also be used to reinforce a plate, should for any reason the screws not be holding well. Where the bone is sloping from large calibre to small, as at the ends of bones, the wire applied thus will slip unless a notch be cut for it to rest in; this need not be very deep.

(3) The ferrule, made of a strip of aluminium or German silver bent round the two ends of the bone is seldom needed, but may be of use where the ends of the fragments are soft and breaking down on attempts to insert screws or wires.

(4) Screws, nails and pegs are of use in fixing on fragments at the ends of bones where there is little room for fixing a plate; moreover there is less dissection needed, for if the fragments be placed in apposition a steel peg may be driven through the smaller into the larger fragment from some other direction than the original incision, after making a small puncture in the skin, and withdrawn again after two or three weeks when some union is present.

Screws, when used singly, *i. e.* without plates to fix on fragments of cancellous bone, *e. g.* the condyles of the femur or an olecranon process, must be long, with coarse thread to bite into the soft bone, and need be threaded only on the half near the point: the carpenter's screw for wood is suitable for such cases.

Plates are chiefly of use in the fixation of fractures of long bones, in which case a long straight plate with four to eight screw-holes is used. Occasionally a triradiate plate is useful to fix a fragment to the end of a bone. The plate should be strong but sufficiently pliable to admit of being bent by the surgeon with pliers, and using a force not gigantic, in order to fit into curved surfaces near the ends of bones. The screws for these plates are quite different from those used singly; as they mostly have to bite in dense bone not more than $\frac{1}{4}$ in. thick at the most, they need not be more than $\frac{1}{2}$ in. long, but the thread must be fine to get a grip in the short amount of bone traversed; the thread should resemble that of screws used for metal-work or rather coarser, and should extend from point to head; the point should be filed off obliquely on two sides so as to cut a thread neatly in the bone. A screw-driver which grips the screw at first but can cast it loose when necessary makes the introduction of screws a much easier matter in difficult places. Care should be taken not to over-twist a screw or the thread will be torn out of the bone and the hold of the screw be lost.

Souttar's girder plate is an ingenious plan to secure great stability with one plate. It consists of a longitudinal plate bent to a right angle along its long axis. After the fragments are placed in apposition a slot is cut longitudinally in the two fragments of bone and one limb of the plate dropped into the slot; the other part then comes against the surface of the bone and is retained with screws, for which holes are provided as in ordinary plates. This girder-formation of the plate renders the bone thus fixed stiff in all directions; it may be more difficult to introduce than are ordinary plates.

October 27th, 1913.

RUPTURED GASTRIC AND DUODENAL ULCERS.

By ANDREW FULLERTON, M.Ch., F.R.C.S.Irel.,

Hon. Surgeon in Charge of Out-patients to the Royal Victoria Hospital and the Belfast Hospital for Sick Children.

THERE are certain cases of acute abdomen which come first under the care of the general practitioner, who must be held responsible for their correct diagnosis and early management. The chief of these are perforations of the hollow viscera, especially the stomach and duodenum, acute appendicitis, strangulated herniæ, acute intussusception (nearly always in children), acute pancreatitis and acute intestinal obstruction. A few hours' delay in any of these cases may lose the patient the chances of recovery which recent advances in surgery have placed within his reach. I propose to devote a few words to the consideration of ruptured gastric and duodenal ulcer inasmuch as in my own practice a large proportion of the acute abdomens, especially in hospital, are due to these accidents. Upon the correct diagnosis of these conditions by the general practitioner the patient is dependent for his life, and it behoves the former to realise his great responsibilities and to know clearly and definitely the signs and symptoms which necessitate the immediate requisition of the services of the surgeon. My experience as a surgeon to a large hospital has taught me that cases are often lost for want of prompt attention, either on the part of the patient or the practitioner in charge. If in doubt it is safer to operate, inasmuch as an exploratory laparotomy in these days is practically devoid of risk.

Let us inquire into the value of previous history in these cases. The classical symptoms of gastric ulcer are pain immediately after taking food, relieved by vomiting, associated with hæmatemesis and melæna. In addition, tenderness on pressure over a definite area in the epigastrium is a well-known sign. When a patient has all these signs and symptoms the diagnosis is easy, but what shall we say about those who suffer from pain in the epigastrium after food and who never vomit and who have no gross signs of hæmorrhage? They may not have even tenderness in the epigastrium. It is a good general rule to keep before one's mind that pain after food of a persistent character is due to some definite pathological condition of the stomach, duodenum, gall-bladder or bile-ducts, or appendix. If the tenderness is to the right of the middle line the duodenum or the gall-bladder ought to be suspected. Pain coming on from one to four hours after food, relieved by food or soda, attended or not with tenderness to the right of the middle line in the epigastric region, is almost certainly due to duodenal ulcer. Again, patients often complain of pain in the back, generally to the inner side of the angle of the scapula. This pain is very frequently associated with a gastric or duodenal ulcer. Or a patient may complain of pains in the chest, under the nipple or along the right or left margin of the sternum. The pains, as I have good reason to know from personal experience, may be due to gastric or duodenal ulcer. Pain radiating round the right lower ribs is a frequent symptom of duodenal ulcer. Gall-bladder dyspepsia resembles to some extent ulcer of the stomach or duodenum. The pain resembles a weight or oppression in the epigastrium, and comes on within half an hour after food and is relieved by belching. Acidity is complained of and the patient is unable to breathe deeply. He suffers from chilliness or gooseflesh.

Attention has been called to these symptoms by Moynihan in his publications on this subject. The same author has recently called attention to what he calls appendix dyspepsia. This is characterised by pain or discomfort after food almost always confined to the epigastrium. Pressure in the iliac fossa will often cause the same sensation of pain or discomfort in the epigastrium as follows usually upon the taking of a meal. The time of its onset after food, however, varies. It may be a few moments, or it may be an hour or two or even more. Flatulence, fulness, and acid, sour belchings are common, and a feeling of intestinal unrest may not seldom be present. Vomiting is often seen, and in some cases hæmatemesis or melæna may occur.

I have had the opportunity of testing the correctness of Moynihan's views in several cases that have come under my care. For example, a lady, æt. about 35 years, had symptoms pointing strongly to gastric ulcer, pain after meals, the pain radiating from the epigastrium to the back. She had also pain under the right lower ribs. On careful examination I found that the patient winced somewhat on

pressure over the appendical region. I explored the stomach and gall-bladder, but found no evidence of disease except a few adhesions extending from below upwards towards the duodenum. I examined the appendix through the original incision and found it kinked about an inch from the tip, which was adherent to a piece of intestine. The removal of the appendix cured the patient and she remains in perfect health. It is now over four years since the operation.

It is a disquieting fact that the first sign of a gastric or duodenal ulcer may be an attack of hæmorrhage or a perforation. I remember seeing a few years ago a powerfully built, healthy-looking policeman who denied ever having had dyspepsia or pain of any kind before his sudden attack. Laparotomy disclosed a ruptured duodenal ulcer.

Are there any prodromal symptoms that would lead one to expect that an ulcer was about to rupture? I have already stated that rupture may occur without any warning whatever. I have also seen cases of rupture where the patient has actually been in bed on the most careful diet. One of my cases was a woman who had fractured her olecranon. She was taken into hospital and the fracture was treated by wiring. A few days later I was called to see her as she had taken a sudden acute pain in her abdomen. I discovered a ruptured duodenal ulcer. During the previous night she had had what she called dyspepsia and had had for many hours nothing but milk. I think that in a good many instances there is some increase of the pain just prior to the rupture, and it is a good plan to warn these patients when they have an exacerbation to keep within reach of help in the event of a perforation occurring. A patient with a known gastric or duodenal ulcer ought not to be sent for a voyage, or allowed to go for long journeys where efficient surgical assistance might not be available. The accident is often caused by some effort such as pushing up a window, reaching up to put clothes on a line, lifting a heavy weight or performing gymnastic exercises. It may also occur after an unusually heavy meal or a meal of unsuitable food. The tenderness over the epigastrium or hypochondrium may be more marked just before rupture.

The first symptom of rupture is a sudden acute pain in the upper abdomen. The pain may be so severe as to merit the description of agonising, so often applied to it by the patient. On the other hand, some patients have actually walked to hospital or to the practitioner after the accident. I operated some years ago on a young man who was employed in one of our engineering firms, who took a sudden acute pain in his abdomen about mid-day while performing some acrobatic feats. He went home and had his dinner and returned to his work as a mechanic and stayed till 5.30 p.m. Later in the evening he walked to the hospital. I operated on him at 11 p.m. the same evening and found a small perforation of the duodenum probably due to an unsuspected ulcer. There was no infiltration round the perforation. He denied any history of previous dyspepsia. There may be a good deal of shock following the onset of the acute pain, but as a rule the patient does not at first give one the idea that he is suffering from a dangerous illness. His pulse may be normal or nearly so, and his temperature is usually subnormal. After a few hours the acute pain generally dulls somewhat and the shock diminishes. It is at this stage that the practitioner may be deceived. The patient does not appear to be collapsed and does not as a rule vomit (I have, however, seen in a few cases frequent vomiting). Under no circumstances should morphia be given. It will mask symptoms, and may produce such a feeling of well-being that the patient will decline operation as unnecessary. I have often had difficulty in satisfying a patient under such circumstances that his life was in danger. There is one sign above all others which should render operation imperative, and that is rigidity. It is a good broad rule to open the abdomen if this sign is present. True, it may occur in other conditions than perforation. It is present when there is intra-peritoneal hæmorrhage, and one sees it occasionally in cases of very severe renal or hepatic colic. It is also, of course, present in inflammatory conditions of the peritoneum. A sudden acute pain in the abdomen followed by rigidity calls for immediate laparotomy. The rigidity may involve one or both recti muscles, and may soon become general so that the abdominal wall becomes board-like.

Following the acute pain in the upper abdomen may be pain in the iliac regions due to gravitation of irritating fluid to the flanks. Loss of liver-dulness is a valuable sign, but it may be absent. I have seen a case with normal liver-dulness while in bed develop resonance after being placed on the ope-

rating table. This was due, no doubt, to movement causing the liver to fall away from the abdominal wall, and possibly to gas being pressed out of the perforation during the transference of the patient from his bed to the operating table. Diminution of liver-dulness may, however, be due to distension of the colon. Fluid in the flanks must be considerable to be recognised by percussion. In the early stage it is entirely absent, and it must not be depended on for diagnosis.

If, then, a patient, with or without a previous history of dyspepsia, takes a sudden severe pain in his upper abdomen and has rigidity of his recti muscles, his abdomen ought to be opened at once. If these cases are operated on without delay the mortality ought to be under 5 per cent. Every hour of delay lessens the patient's chances. At the Royal Victoria Hospital we do not wait for any shock that may be present to pass off. The anæsthetic actually seems to improve the condition of the patient. The abdomen may be opened to the right of the middle line and the rectus displaced outwards in order to avoid a ventral hernia. These abdomens are often very hard to sew up, especially in powerful muscular men with great rigidity. The posterior layer of the sheath of the rectus tears away with the sutures unless a good bite be taken and the edges gently drawn together. A good assistant is invaluable here. When the peritoneum is incised gas may escape and turbid fluid. Sometimes the contents of the stomach have extravasated widely. Thus, tea-leaves, currants, fruit skins and porridge are not unfrequently seen in the peritoneal cavity. On the other hand, one often finds no food *débris*, simply a turbid fluid, which is oily looking if the duodenum be the seat of the perforation. The perforation may be hard to find. The ulcer may be on the anterior surface of the duodenum or pyloric end of the stomach. In such cases, unless there are dense adhesions, no great difficulty will be experienced, but when the perforation is at the cardiac end of the stomach or posteriorly it may be very troublesome to locate with fluid welling up into the wound. If the perforation is not found anteriorly the stomach must be turned up and the transverse mesocolon torn through to reach the small sac. The perforation is usually circular, surrounded in most cases by a thickened, œdematous, friable area often difficult to suture. The sutures tend to cut through the thickened tissue as if it were a piece of turnip, and much care must be exercised to bring the margins of the opening together. Sometimes a purse-string suture answers best. I always use linen thread, as I am afraid that catgut may not be sufficiently durable. Several layers of interrupted or continuous sutures may be required before the opening is secure. It is well to bring a piece of omentum up over the sutured area, and stitch it in position so as to afford additional security against leakage. If the extravasated fluid is small in amount I mop it up gently and close the abdomen without drainage. If there is a considerable amount of fluid present I place a rubber tube in Douglas's pouch and close the abdomen without lavage. I am sure I have lost patients by prolonged irrigation, and I never now irrigate. If I see any foreign particles in the peritoneal cavity I remove them as far as possible with a gauze wipe. The rest are left to come away through the tube. I have no doubt foreign matter is often left in the abdomen. What happens to it I do not know, but the patients recover. Rapidity of operation is essential, and the patient must be got back to bed as soon as possible. Warm bottles are applied, and he is placed in the Fowler position. Saline, either by continuous irrigation *per rectum*, or by two- or four-hourly enemata, is administered.

I have carefully weighed the question of immediate gastro-enterostomy in cases of ruptured duodenal or pyloric ulcer, and have carried it out successfully in several cases. I have, however, recently seen a very promising case operated on within a few hours die as a result of combining the two operations at one sitting. Unless the surgeon is a rapid operator it is unwise to do more than get the patient over his immediate danger. Possibly he may never require gastro-enterostomy. If his symptoms persist the operation may be carried out later under more favourable conditions. I have in cases of small ulcers run the knife round the edge so as to remove it prior to suture, much as one might remove the "eyes" from a potato, but further than this I am not with my present experience prepared to go. Of course, if the suture narrows the outlet from the stomach to a marked degree it is wise to short-circuit if the state of the patient will permit.

As to the after-treatment, surgeons differ very much in their methods. Fluids at first, followed very tentatively by such things as Benger's food, jellies, etc., are allowed. For the first twenty-four hours it is probably better to confine the patient to sips of hot water. There is always some anxiety about the bowels. If the ulcer has been well sutured saline aperients are not likely to do harm. Distended intestines must be emptied, and personally I have no hesitation in an ordinary case of medium severity in administering after twenty-four hours sulphate of magnesium with this object in view. If the intestines show signs of commencing paralysis at the time of operation, small doses of magnesium sulphate at frequent intervals may be almost immediately started in the hope of relieving the distension.

Even when all seems to be going well, the patient may die from various causes other than shock or peritonitis. I have had deaths from subphrenic abscess, pulmonary embolism, hæmatemesis (on the fifteenth day from a second ulcer), and persistent vomiting after four weeks probably due to adhesions. It would be interesting to discover how many lives have been saved since the introduction of surgical intervention in cases of ruptured gastric and duodenal ulcer. The mortality of these accidents is still steadily diminishing, and will diminish in direct ratio with the promptness with which they are recognised and treated.

October 27th, 1913.

GLYCOSURIA AND ALLIED CONDITIONS. By P. J. CAMMIDGE, M.D. Pp. 458. London: Edward Arnold, 1913. Price 16*s*. net.

Dr. Cammidge is well known as an enthusiastic worker in chemical pathology, especially in connection with glycosuria and diseases of the pancreas. His conclusions have aroused wide-spread interest, which has stimulated many to observe phenomena that would otherwise have escaped them. His insistence on the chemical problems of disease is useful at a time when the tendency is to concentrate, perhaps too much, on bacteriology. The present work includes an account of the chemical properties of the carbohydrates and their derivatives, which is sufficiently full to be of value for reference. He then approaches glycosuria from the experimental side, showing how light can thus be thrown on the problems of diabetes. Then follows a chapter on transitory glycosuria, which will repay careful reading. The various aspects of persistent glycosuria are next considered. Like other modern workers, Dr. Cammidge refuses to draw a hard and fast line between the slighter forms of glycosuria and diabetes, as all intermediate grades exist. At present, when so much is being said about internal secretions, his remarks on the influence of the ductless glands on carbohydrate metabolism are of special value. The author insists on the quantitative as well as qualitative regulation of the diet, and his instructions do not err on the side of simplicity. Like Von Noorden, he finds that some diabetics are more sensitive to animal proteins than to carbohydrates. The book is an important contribution to the literature of the subject by an original worker.

IONIC MEDICATION: THE PRINCIPLES OF THE METHOD AND AN ACCOUNT OF THE CLINICAL RESULTS OBTAINED. By H. LEWIS JONES, M.D., F.R.C.P., Consulting Medical Officer to the Electrical Department in St. Bartholomew's Hospital. Pp. 151. Crown 8vo. London: H. K. Lewis, 1913. Price 5*s*. net.

In these days when so many books are published which are entirely superfluous—not to say worse—it is refreshing to the jaded reviewer to meet one such as this which emphatically supplies a want. Ionic medication is one of the new methods of treatment which are a distinct advance, and no one in this country is better qualified to describe it than the author, who is one of its pioneers. The laws of electrolytic conduction and their application are briefly explained. The use of the various ions, zinc, copper, mercury, silver, lithium, magnesium, radium, alkaloidal, salicylic, chlorine and iodine ions is described. The best results have been obtained in superficial affections, such as ulcers, urethritis, vaginitis and endometritis. In rheumatoid arthritis Dr. Jones thinks that ionisation is beneficial in cases associated with pyorrhœa. The book can be recommended as an excellent guide.

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SOME DISTURBANCES OF THE SINGING VOICE.*

By DUNDAS GRANT, M.A., M.D., F.R.C.S.,
Hon. Surgeon, Royal Academy of Music and Royal Society of Musicians; Consulting Surgeon, Central London Ear and Throat Hospital, etc.

SINGING as an accomplishment may be a source of immense pleasure both to the singer and the hearer, but as a profession it is full of anxiety, for its successful practice absolutely depends on the healthy condition of the vocal organs. In a family party it is not a matter of great importance if those who can sing won't and those who can't sing, will, but the public is not so tolerant and withholds its rewards from those who fail to come up to the required standard.

It is not my purpose to give you an exhaustive account of the disturbances to which the singing voice is liable, as there is no lack of books purporting to supply this and to give the methods of cure which the various authors have found of the greatest value. The confidence with which each extols his own methods is not less striking than the emphasis with which he decries those of others. The inference is that "all are right and all are wrong." I mean to say, that in every system or method there is much that is good for certain cases but not by any means for every case.

I shall leave for another occasion the critical consideration of the methods for which claims have been made, and shall content myself with giving you an account of a series of cases which have come under my observation. I have examined them without bias in the direction of any special teachings, and shall endeavour to report to you the conclusions to which I have been led in the light of the vocal and physical signs and of the results of the treatment prescribed.

* Lecture delivered at the Polyclinic.

The disabilities described by the patients are given as far as possible in their own words. In the following tables the numbers show the relative frequency of the various symptoms in 131 cases of singers who have applied to me for treatment of their vocal organs. They might be classified under the headings of "Defective Vocal Utterance" (Table I), and "Abnormal Sensations associated with Singing" (Table VIII).

Defective Vocal Utterance.

The requirements in the singing voice are sweetness of tone, clearness of resonance, volume, flexibility, reliability of intonation and musical ear, accompanied by ease and comfort in function.

Departures from these may take the form of *defective vocal tone*, such as hoarseness, huskiness, roughness, exaggerated break, difficulty with "head" tone; *defective vocal power* expressed as loss of singing voice, weakness, unreliability, thinness, etc.; *impaired resonance*, "thickness," veiling or mistiness, woolliness; *nasal tone; faulty intonation* (Table I).

Table I.

Defective Voice Utterance	88
Hoarseness or huskiness	39
Roughness of voice	3
Grating voice	1
Roughness of low tones	1
Reediness	1
Break exaggerated between "B" and "C"	1
Difficulty with head notes	5
Loss of voice	6
Weakness of voice	4
Unreliableness in singing	2
Thinness of voice	1
Want of resonance	8
Thickness of voice	7
Veiling or mistiness	4
Woolliness	1
Nasal tone	3
Faulty intonation	1

Of the 39 who complained of *hoarseness or huskiness*, out of 37 of whom notes are available, 21 had obvious changes in the larynx, such as the presence of mucus on the vocal cords, slight inter-arytenoid swelling, inflammatory swelling of the cords with defective adduction, congestion of the cords, maceration of the cords, nodulation of slight degree, crust-formations in the trachea, slight subglottic thickening, thickening of vocal process, paresis of the internal tensors (Table II).

Table II.

Conditions found in 21 Complaining of "Hoarseness or Huskiness" with obvious Changes in Larynx.

Congestion of vocal cords	5
" " with slight nodulation	2
" " with hypertrophic rhinitis	1
" " adenoids	1
Mucus on vocal cords	4
" " with hypertrophic rhinitis	2
" " with congestion of larynx	1
Interarytenoid swelling	3
" " with slight nodulation	1
" " with slight adenoids	1
Nodulation of vocal cords	1
Inflammatory swelling of vocal cords with defective adduction	1
Maceration of vocal cords with hypertrophic rhinitis	1
Slight subglottic thickening and weakness of palate	1
Subcordal thickening and interarytenoid thickening	1
Infiltration below vocal cords and paresis of internal tensors	1
Thickening of vocal process and adenoids	1
Swelling of arytenoids and adenoids	1
Crust formation in trachea and enlargement of lingual tonsil	1

Similar in kind are the complaints of *roughness of the voice*, and the conditions found were congestion of the vocal cords, catarrh in the trachea, congestive laryngitis; *grating voice*, and *roughness of low tones*, in both of which there is no report about the larynx; *reediness*, in which there were inflammatory changes in the larynx; *break exaggerated between B and C*, in which there were chronic rhinitis and weakness of the internal tensors (Table IV).

In sixteen in which there were no definite laryngeal changes we find such conditions as imperfect action of the palate, deflection of the nasal septum, granular pharyngitis, septal spur and hypertrophic rhinitis, adenoids and elongated uvula, adenoids, muco-pus in the naso-pharynx, enlargement of the lingual tonsil, general debility (Table III). There is little doubt that in some of these cases there was some slight degree of laryngeal involvement, although insignificant in comparison with the associated changes in the nose or naso-pharynx.

Table III.

Conditions found in 16 Complaining of "Hoarseness or Huskiness" without obvious Changes in Larynx.

Adenoids	5
Muco-pus in naso-pharynx	1
Adenoids and elongated uvula	1
Deflection of the nasal septum	1
Septal spur and hypertrophic rhinitis	1
Granular pharyngitis	1
Imperfect action of the palate	1
Enlargement of the lingual tonsil	2
General debility	3

In those complaining of *difficulty with head notes*, the conditions found were roughness of the vocal cords with enlarged tonsils, paresis of the internal tensors with exophthalmic goitre, enlargement of the lingual tonsil and hypertrophic rhinitis, normal larynx (the patient being a lady of "masculine" type), and normal larynx (Table IV).

TABLE IV.

Conditions found in—

3 *Complaining of "Roughness of the Voice."*

Congestion of vocal cords	1
Catarrh in the trachea	1
Congestive laryngitis	1

1 *Complaining of "Grating Voice."*

Hypertrophic rhinitis and septal spur	1

1 *Complaining of "Roughness of Low Tones."*

Hypertrophic rhinitis and septal deviation	1

1 *Complaining of "Reediness of Voice."*

Inflammatory changes in larynx	1

1 *Complaining of "Exaggerated Break between 'B' and 'C.'"*

Chronic rhinitis and weakness of internal tensors	1

5 *Complaining of "Difficulty with Head Notes."*

Roughness of vocal cords with enlarged tonsils	1
Paresis of internal tensors with exophthalmic goitre	1
Enlargement of lingual tonsil with hypertrophic rhinitis	1
Normal larynx (patient of "masculine" type)	1
Normal larynx	1

In those characterised mainly by *weakness*, amounting in some cases almost to *loss of voice*, such conditions were found as general debility, congestion of the vocal cords, slight nodulation, imperfect approximation of the cords (Table V). The association of the symptom with the conditions observed is in this group pretty obvious.

TABLE V.

Conditions found in—

6 *Complaining of "Loss of Voice."*

General debility	3
Congestion of vocal cords	2
,, with thickness of epiglottis	1
(No report in the sixth)	

4 *Complaining of Weakness of Voice.*

Congestion of vocal cords with slight nodulation	1
Distinct nodules	1
Imperfect approximation of cords with nasal spur	1
General debility with normal condition of larynx	1

2 *Complaining of "Unreliableness in Singing."*

Defective approximation of the vocal cords	1
Anæmia	1

1 *Complaining of "Thinness of the Voice."*

General constitutional weakness and chronic rhinitis	1

It will be seen that there is generally some obvious laryngeal condition with physical changes of sufficient importance to disturb the vibrations of the vocal cords (and column of air), so accounting for the want of clearness and pleasantness of the laryngeal tone. At the same time such conditions are frequently conspicuous by their absence, and the cause of the disability has to be sought elsewhere in the complex organisation which makes up a singer.

Want of resonance, thickness of voice, veiling or mistiness, woolliness, were found in a group in which the complaint, as will be seen, was not described as "hoarseness" or "huskiness" but as a vague disturbance in the quality of the tone. There were generally found conditions which produced some nasal obstruction, such as hypertrophic rhinitis, adenoids, enlarged tonsils, septal spur. These occasionally produced a nasal tone, generally of the character of rhinolalia clausa or the voice of the obstructed nose, and occasionally rhinolalia aperta or the voice of the defective palate, or a combination of the two in one case in which there was post-diphtheritic weakness of the palate combined with posterior turbinal hypertrophy (Tables VI and VII).

TABLE VI.

Conditions found in—

8 *Complaining of "Want of Resonance."*

Hypertrophic rhinitis	1
Adenoids	1
,, with enlarged tonsils	1
,, with septal spur and slight congestion of larynx	1
,, with posterior hypertrophy of turbinated bodies	1
Septal spur	1
,, with slight amount of adenoids	1
Maceration of cordal mucous membrane (repeated colds)	1

7 *Complaining of "Thickness of Voice."*

Adenoids	2
,, and slight nasal spur	1
,, and "elliptical" larynx	1
Septal spur and hypertrophic rhinitis (with anæmia)	1
Congestion of larynx (post-influenzal)	1
Normal larynx	1

4 *Complaining of "Veiling or Mistiness of Voice."*

Adenoids	1
,, with enlarged tonsils and enlarged lingual tonsil	1
,, and weakness of internal tensors	1
Simple chronic rhinitis and naso-pharyngeal catarrh	1

1 *Complaining of "Woolliness of Voice."*

Septal deflection and hypertrophic rhinitis	1

TABLE VII.

Conditions found in—	
3 *Complaining of "Nasal Tone."*	
Septal spur and enlarged lingual tonsil	1
,, and adenoids	1
Weakness of palate (post-diphtheritic) and posterior turbinal hypertrophy	1
1 *Complaining of "Faulty Intonation."*	
Adenoids and enlarged tonsils (normal larynx)	1

Faulty intonation (Table VII), or singing out of tune, was complained of in only one of the cases under my observation. The local condition found was adenoids and enlarged tonsils (the larynx being normal). I regret that my notes of this case are imperfect as regards the nature of the faulty intonation, but I do not consider the local conditions found are likely to have accounted for it.

What causes defective ear for tune is very difficult to say, but it seems to be innate in some people. If slight in degree and with a tendency to flatness, it is apt to be present only when the patient is suffering from some intercurrent catarrh or general debility and is of very hopeful prognosis with judicious training and attention to general health. The case here noted was to the best of my recollection of this character.

If, on the other hand, the patient sings "sharp" the prognosis is much less hopeful. I have known a few cases of marked defect of ear in singers greatly improved by learning a string instrument such as the violin or viola, even if only in a very elementary degree. Of course, this calls for an effort of attention and study, and people with "voices" are not always inclined to make such efforts.

ABNORMAL SENSATIONS ASSOCIATED WITH SINGING.

In forty-three cases the complaint was of such a symptom as *aching on singing*, *tired feeling*, *soreness*, *feeling of swelling*, etc. (Table VIII).

TABLE VIII.

ABNORMAL SENSATIONS ASSOCIATED WITH SINGING	43
Aching on singing	11
Tired feeling	8
Soreness of the throat	6
Feeling of swelling	6
Difficulty in singing	4
Cough	4
Relaxed throat (soreness)	1
Tightness	1
Dryness	1
Irritation (tickling)	1

In those with *aching* or allied sensations, we find enlarged tonsils (sometimes with adenoids), lateral pharyngitis, chronic rhinitis, relaxation of vocal cords, "powerful" voice out of proportion to the delicacy of the larynx, and in more than one case a completely normal larynx (Table IX).

In the majority of the cases there was some inflammatory condition in the upper part of the throat. Of the remainder, one was explicable by the strain on the muscles of the neck associated with relaxation of the vocal cords, another by general nervous hyperæsthesia. In the last on the list the disproportion between the force of the expiratory blast and the delicate structure of the larynx and vocal cords was remarkable. The patient was over-impressed with the idea that her voice was pre-eminently a "powerful" one, as indeed it was, and she used it as such, but the vocal cords were of too delicate construction to stand the strain without suffering, and in point of fact there developed an inflammatory swelling on the under surface of the cords. She was encouraged to practise, we might almost say to affect refinement and softness of singing voice, as contrasted with the somewhat proletarian loudness to which she had been addicted. Her teacher has found an improvement already.

In those complaining of a *tired feeling*, the physical changes were very slight or none, and there was usually marked general debility (Table IX). In the case with slight swelling of the ary-epiglottic folds and imperfect adduction of the cords, considerable improvement followed the use of massage to the neck and applications of a solution of chloride of zinc to the larynx.

Soreness of the throat was generally found associated with adenoids, laryngeal catarrh, enlarged tonsils or enlarged ligual tonsil (Table IX).

With a *feeling of swelling*, as will be seen by the table (Table IX), there were present enlargement of tonsils, enlargement of lingual tonsil, naso-pharyngeal catarrh; in these cases there was generally considerable nervous rather than physical change.

Difficulty in singing occurred with slight nodulation of the cords or congestion with adherent mucus (Table IX). *Cough* was present in a few cases in which there was found mucus on the vocal cords or in the trachea, enlarged lingual tonsil, adenoids and post-influenzal cough (Table IX).

In a case in which *relaxed throat* was complained of there was found simply turgescence of the

inferior turbinals, relief following the use of the galvano-cautery. *Tightness* was complained of in a case of enlarged lingual tonsil, removal of which brought about the disappearance of this feeling (Table IX). In a case of *dryness* there was hypertrophic rhinitis, and with *irritation in the throat*, enlarged tonsils, adenoids and enlarged lingual tonsil (Table IX).

TABLE IX.

Conditions found in 11 *Complaining of "Aching on Singing."*

Enlarged tonsils and adenoids	2
Enlarged tonsils	1
Chronic lacunar tonsillitis	1
Slight adenoids	1
Slight turgescence of arytenoids and lateral pharyngitis (inflammatory)	1
Chronic rhinitis (thickening on floor of nose)	1
Relaxation of vocal cords (muscular pain)	1
Normal throat (hyperæsthesia)	2
"Powerful" voice, small larynx	1

8 *Complaining of "Tired Feeling."*

Slight laryngeal congestion	1
Slight swelling of ary-epiglottic folds, imperfect adduction, and slight adenoids	1
Enlarged lingual tonsil	2
Adenoids	2
Normal throat	2

6 *Complaining of "Soreness of the Throat."*

Adenoiditis with catarrh of larynx (mucus on vocal cords)	1
Posterior turbinal hypertrophy	1
Enlarged tonsils with hypertrophic rhinitis	1
Enlarged tonsils, adenoids, and enlarged lingual tonsil	1
Enlarged lingual tonsil	1
Weakness of internal tensors	1

6 *Complaining of "A Feeling of Swelling."*

Enlargement of tonsils	1
Enlargement of tonsils and enlarged lingual tonsil	1
Enlarged lingual tonsil and thyroid gland	1
Naso-pharyngeal catarrh	1
Turgescence of turbinals, slight thickening of left vocal cord	1
Normal throat	1

4 *Complaining of "Difficulty in Singing."*

Slight nodulation	1
Congestion with adherent mucus on cords	1
Colds	1
Normal throat	1

4 *Complaining of "Cough."*

Slight nodulation and mucus on vocal cords, slight thyroid fulness	1
Mucus in trachea, enlargement of lingual tonsil	1
Adenoids	1
Normal larynx, post-influenzal cough	1

1 *Complaining of "Relaxed Throat."*

Turgescence of the turbinated bodies	1

1 *Complaining of "Tightness."*

Enlarged lingual tonsil	1

1 *Complaining of "Dryness."*

Hypertrophic rhinitis	1

1 *Complaining of "Irritation in the Throat."*

Enlarged tonsils and adenoids with enlargement of lingual tonsil	1

GENERAL REVIEW.

Definite laryngeal changes were found in 62 (Table X), enlargement of the lingual tonsil in 15 (Table X), nasal disease in 31, naso-pharyngeal and pharyngeal disease in 47 (Table XI), and general and miscellaneous morbid conditions in 17 (Table XII).

TABLE X.

Changes found in the Larynx.

Nodulation of vocal cords	8
Congestion of vocal cords	12
„ of the larynx in general	5
Mucus on the cords or in the trachea (generally with hypertrophic rhinitis or post-nasal catarrh)	10
Laryngitis as such	1
Maceration of mucous membrane of vocal cords	2
Subglottic infiltration	3
Thick epiglottis (normal)	1
Slight fulness of arytenoids	2
„ swelling of ary-epiglottic folds	1
Interarytenoid swelling	4
Slight fulness of left vocal process	1
Weakness of internal tensors	10
Smallness of larynx	1
Roughness of vocal cords	1
Enlargement of the lingual tonsil (Removed in 7 cases; report of great improvement in 6; no report in 1.)	15

TABLE XI.

Changes found in Nose, Naso-pharynx and Pharynx.

Hypertrophic rhinitis (In 5 there was slight deflection of septum as well, in 7 galvano-cautery puncture was performed.)	19
Septal spur (removed in 1)	7
Septal deflection (marked)	1
Chronic rhinitis	3
Inflammatory thickening of floor of nose	1
Adenoids (removed in 13)	26
„ with enlarged tonsils (removed in 3)	6
Enlarged tonsils (removed in 2, punched in 1)	5
Naso-pharyngeal catarrh	4
Imperfect action of palate	2
Lateral pharyngitis	1
Granular pharyngitis (marked)	1
Lacunar tonsillitis	1
Elongated uvula	1

TABLE XII.

General and Miscellaneous Conditions.

Enlarged thyroid body	2
Exophthalmic goitre	1
General weakness	10
Heart disease	1
Anæmia (marked)	1
Post-diphtheritic weakness of palate	1
Masculinity of type in a female	1

It will be seen that in some cases there were positive physical conditions, some significant and some quite insignificant; in other cases no physical changes detectable. As a rule the association between the special symptom and the special physical changes was fairly clear, but in a number of instances it was far from obvious.

It must be remembered that a singer is not merely a vocal organ but a complex human organism, requiring a healthy and vigorous vocal organ, a normal and vigorous body and a highly sensitive nervous system. All these are of importance to him in view of the fact of his livelihood and reputation depending upon them, and they have to be taken into account in the carrying out of the treatment to be described.

(*To be continued.*)

November 3rd, 1913.

TABULAR DIAGNOSIS. By R. W. LEFTWICH, M.D., late Assistant Physician to the East London Children's Hospital. London: Edward Arnold. *7s. 6d.* net.

This book might correctly be described as a time-saver for the busy and overworked practitioner. The author had previously done a like service to to the profession in his 'Index of Symptoms.' This book consists of a collection of tables—350 in all—each table in three columns. In the first is stated the symptom organ or region, while the word or phrase which qualifies it is placed in the other two columns. In this way each table shows a contrast. For example, bulbar paralysis with tumour at base of brain, minor epilepsy with syncope, and so on. At the foot of the page the symptoms which the contrasted diseases have in common are stated. The book expresses differential diagnosis at a glance, and in this age of pressure and hurry will supply a want, especially, shall we say, to the "panel" doctor.

THE MODERN TREATMENT OF HEART-DISEASE IN CHILDREN.*

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THE chief recent improvement in the treatment of cardiac affections in children, apart from the introduction of a few new drugs, depends on the recognition of the frequency of myocarditis and its effects as the fundamental factor in the majority of cases in children. In this respect our knowledge has advanced, and in consequence we have learnt to appreciate the necessity for prolonged rest. Important points for discussion are the limits which should be placed on the duration of rest and the subsequent use of exercises; the value of blistering, leeches and venesection in acute inflammations and severe dilatation; the use of digitalis and its products; the value of special purgatives and diuretics; and the indications for operative treatment.

To a certain extent the methods of treatment are modified by age, for the child is a very restless animal, its metabolism is active, and the heart is a growing organ. Fortunately the degenerative changes consequent on age, alcohol, syphilis and renal diseases are very rare. During periods of rapid development, especially at puberty, the cardiac growth may not be proportionate to that of the body, and the heart may become functionally incompetent, as indicated by undue frequency of the pulse and shortness of breath. Functional disorders of the heart are comparatively uncommon in children. In many a so-called functional case there is a primary myocardial affection, the result of latent rheumatism or the sequel of some infective disorder. It is apt to follow influenza, is a frequent sequel of diphtheria, and may even ensue on varicella or the common influenza cold. Obviously in such cases successful treatment depends on accurate diagnosis. It is likely to prove unsatisfactory if slight myocardial mischief is regarded as functional nervous disturbance. And it must be

* Read at the Royal Society of Medicine.

remembered that a cardiac murmur does not always mean valvular defect, nor does the absence of a murmur necessarily exclude serious disease.

In applying treatment we must take into consideration the nature of the mischief and the valves affected, if any; the nutritional and functional powers of the cardiac muscle; the idiosyncrasy of the patient and the irresponsibility of childhood; the environment of the child, and the difficulty in securing parental co-operation as soon as the obviously troublesome symptoms have subsided.

Rest is the most important factor in treatment. It is not needed if there is good compensation; then, it is only necessary to guard against overstrain. The duration of rest depends on the degree of dilatation consequent on myocardial incompetence, and on the existence of endocarditis, myocarditis or pericarditis alone or in combination. It may be impossible to ascertain whether a mitral systolic murmur, associated with cardiac dilatation, is due to endocarditis or to incompetence of the valvular orifice because of dilatation and myocarditis. Rest must be continued for one to three months after all acute symptoms have subsided and compensation has been established. It must be most prolonged in myocarditis. Special care is needed during rapid growth and at puberty, periods at which cardiac strain must be carefully avoided, if there is the least suspicion of previous mischief or if there is any functional incompetence. It is a mode of treatment easily carried out in young and placid children, and perhaps extremely difficult in excitable, vigorous children over ten years of age. A trained nurse is almost invariably essential. In the early stages rest must be absolute, and every cause of excitement, such as visitors, new toys and games, must be prohibited. As compensation is acquired rest is gradually replaced by massage, passive and active movements, and exercises. A warning is necessary against the abuse of exercises for children with even well-compensated cardiac disease. The modern craze of flying to Swedish and other exercises has been, in my experience, responsible for the cardiac breakdown of several children. The growing heart, weakened by disease, is easily over-strained. Exercise must not cause dyspnœa or anginal pain. For three to twelve months it is limited to walking on level ground, and is then increased successively by minor gymnastics, hill-climbing, bicycling on the level, and appropriate games.

Baths, saline and carbonated, such as given at Nauheim and also in this country, and electrotherapeutic methods of treatment, are perhaps unnecessary in childhood. Baths stimulate the cutaneous nerves, dilate the capillaries of the skin and slow the action of the heart, exciting more vigorous action, and diminish the cardiac dulness. They are suitable for moderate cardiac dilatation and can be given on alternate days, with Schott's exercises on the other days.

Diet.—In acute cases, especially if rheumatic in origin, a diet of milk and carbohydrates is advisable. Some physicians allow chicken broth, beef tea, and other meat extracts. Personally, I regard such extracts as unsuitable, and prefer to order fruit or vegetable soups, made without meat stock, as a change and to counteract constipation. In all cardiac affections the food should be nutritious, easily digestible, and given in small meals so as to avoid overloading and flatulent distension of the stomach. The state of the appetite is a valuable indication of progress. One of the first signs of failing compensation in chronic heart disease is anorexia after exercise. It is followed by dyspepsia, impaired nutrition, restlessness at night or insomnia, anæmia, cough, shortness of breath, and swelling of the legs. One of the most reliable signs of improvement is gain in weight, provided there is no œdema. In cases of œdema a salt-free diet is sometimes advantageous, combined with a moderate limitation of fluids. It is not necessary to prescribe a strict diet in chronic cardiac failure, provided that the food is nutritious, given in small meals and palatable. There is too great a tendency to order milk in quantities larger than the patient can digest efficiently, with the result that the appetite becomes impaired, the tongue furred and the breath offensive, the stomach dilated and the bowels constipated.

Blistering is in my opinion of very doubtful value in endocarditis and myocarditis. That a blister on the skin can affect an inflammation of the cardiac valves requires a more optimistic imagination than I dare own to. Although such treatment is used and highly recommended, it is associated with other remedies which are more probably the cause of the favourable issue. In pericarditis blistering seems undoubtedly beneficial. The best

method of application is the use of flying blisters around the inflamed area. Other counter-irritants may be used.

The local application of *ice-bags* in the treatment of cardiac inflammations has been warmly advocated, especially for pericarditis. Sometimes it appears to quiet the excited action of the heart. Possibly this result is merely due to the complete rest so necessary for the accurate adjustment of the treatment. As a rule children do not like cold applications. Poultices and fomentations are more soothing and can do no harm. The addition of opiates or belladonna does not make them more efficacious.

Venesection and *leeches* must be used with caution, for children stand the loss of blood rather badly. If there is much pain, an acute pericarditis, or an acute dilatation of the right ventricle due to myocarditis or cardiac failure, the application of a few leeches over the liver affords great relief. Venesection to the extent of a few ounces may be essential. Bleeding relieves the distension of the right heart for some hours and allows time for other remedies to exert their effect. It must not be adopted lightly, and it ought not to be allowed to fall entirely into disuse. It should be looked upon as a measure for immediate relief of the straining heart and not as a means of reducing inflammation.

Purgatives are essential. It is important to keep the bowels open, and freely so if there is backward pressure and a tendency to œdema. If the liver is engorged there is no pill as valuable as that of mercury, squill and digitalis, given at bedtime and followed by a saline cathartic in the morning. Such a pill may be given twice or three times a day. In cardiac inflammation the hydragogue cathartics are the most valuable purgatives, for they relieve the congestion of the tissues throughout the body and reduce the work required of the heart. If purgatives are required in cases of compensated chronic heart disease, the various preparations of paraffin, senna, aloes or cascara may be used. A weekly dose of calomel or blue pill is sometimes advantageous.

Diuretics are also of immense value in acute inflammations and in the relief of œdema. Their action can be assisted by dry-cupping over the kidneys. Dry-cupping over the bases of the lungs is useful in passive congestion of these organs. I have no experience of the tincture of cantharides recommended by Dr. Eustace Smith as a diuretic, nor am I prepared to state which is the best diuretic. Diuretin, caffeine and theocin sodium acetate are all of value, the last mentioned often increasing the efficacy of digitalis.

Digitalis is a diuretic and a cardiac tonic. There is a tendency to limit its use to non-inflammatory cardiac affections, though it may be prescribed tentatively in the course of inflammatory ones, with the exception of endocarditis. It is not always required because a murmur is present. Formerly the infusion was preferred. Herbalists recommend that the leaves should be gathered at night, and there is recent evidence that they contain more of the active principle at this time. Now, most physicians prescribe the tincture as being more stable and regular in composition. It is the simplest and most available preparation. The drug should be given every six hours until a definite reaction is obtained, and repeated if relapse occurs. Try to find the dose which maintains improvement without causing unpleasant symptoms. The patient's own sensations are a good guide. The drug must be omitted, if it decreases or does not increase the secretion of urine; if it causes considerable slowing of the pulse; if it gives rise to anorexia, nausea, and confusion of thought; and if the patient cannot be kept under supervision. Frequently, continuous administration is advisable, less often in children than in adults, and chiefly for cardiac failure due to mitral regurgitation. For such cases, after compensation is fairly well established, a dose of the powdered leaves, $\frac{1}{2}$ to 2 gr. once a week or more often, will maintain the compensation. Or the drug may be given for three to four days at a time every month. I have obtained excellent results both in adults and children by such means, using Nativelle's crystallised digitaline in doses of $\frac{1}{10}$ to $\frac{1}{4}$ mgrm. daily for a week at a time. Digitalone (Parke, Davis & Co.), is physiologically standardised. Ten minims are equivalent to eight of the tincture and to 1 gr. of the leaves. Digalen, a sterile amorphous digitoxin, is said not to be cumulative. This has been disproved recently. One cubic centimetre is equivalent to 0·3 mgrm. digitoxin, folia 2·05 gr., tincture 18 minims, and infusion 5·4 drm. These drugs can be given subcutaneously and by rectum if necessary. Crystalline digitaline is official in the French Codex. It corresponds to the German digitoxin, and is much more powerful than amorphous digitaline.

Most of these preparations are recommended because of their constant strength, and it is possible that the claim may be true, and also on the grounds that they are non-cumulative and less apt to upset the stomach. In some cases strophanthus is more suitable—*e. g.* in severe dilatation and mitral stenosis. Squill, sparteine sulphate, strychnine and nux vomica are also valuable. Alcohol is necessary in acute dilatation, myocarditis, and cardiac failure. Cardiac stimulants are not needed if recumbency alone is sufficient to reduce the action of the heart to the normal rate and if compensation is established. They are most efficacious when the right side of the heart has been relieved by cupping, leeches, or venesection, and the liver relieved by mercury and a saline aperient. I know nothing about the tincture of cereus mexicana, which has been recommended, in doses of 5 to 30 minims for adults, to increase the strength and regularity of the heart without raising the blood-pressure. Vaso-dilators are unnecessary, for blood-pressure is almost invariably low in children. Perhaps iodides are of value in chronic pericarditis.

Salicylates, and their congeners, are generally prescribed in all cardiac inflammations of rheumatic origin. The remarkable effect on the temperature, joint pains and swelling of rheumatic fever is strong evidence of the great value of this drug. Yet, although I have used it systematically for a quarter of a century, I still find it difficult to advance reliable evidence that the drug affects the course of cardiac inflammation. In my experience it is by no means uncommon for the myocarditis to run a prolonged course with mild fever, the temperature running up to about 100° F. at night, in spite of the liberal administration of salicylates or aspirin.

Summary.

In all inflammatory cases in which there is the least suspicion of rheumatism salicylates and alkalies must be prescribed. The patient is kept in bed on a light milk and carbohydrate diet, fruit and vegetable soups being allowed as a change. Complete rest and a trained nurse are necessary. Blistering or counter-irritants may be used, but are chiefly efficacious in pericarditis. Vaccines and serums are of problematic value. There is hope that, when the organism of rheumatic fever is isolated, an autogenous vaccine may prove beneficial. I do not wish to ignore the extremely valuable work done in this connection, but, though realising that many observers are agreed that the genuine organism has been already discovered, there seems to be at present a disinclination to accept it as absolutely proved. In infective endocarditis—fortunately infrequent in children—my experience with vaccines and serums has proved unsatisfactory. It is difficult to grow the incriminated organism from the blood, difficult to prove that the organism obtained in cultures is the one causing the disease, and difficult to obtain good results from an autogenous vaccine or from a polyvalent serum.

Recourse is sometimes had to paracentesis for the relief of pericardial effusion. It is rarely essential in rheumatic pericarditis, for even the largest effusions may be absorbed. It is a point for discussion whether the operation would promote more rapid recovery. For purulent effusions the pericardium must be opened and drained. Another and more modern operation, sometimes called cardiolysis, is resection of the rib cartilages over the cardiac area, for the relief of general adhesion of the pericardium. The treatment of cardiac failure and backward pressure is the same at all ages. For extreme œdema of the lower limbs multiple punctures and sterilised dressings are more satisfactory than the insertion of Southey's tubes. Iron and glycerophosphates are given when compensation is established. Compensation is maintained by regulation of the mode of life, diet, sleep, hygiene and exercise. These patients must not be allowed to become chronic invalids. A more just appreciation of their capabilities is now general in comparison with the views held by the past generation. If there is merely some mitral regurgitation, the only precautions necessary are against rheumatism and overstrain. Such children ought not to take part in football, races, steeplechases, and like competitions. They may take part in other varieties of exercise, during which they can stop as soon as they feel any cardiac discomfort. If compensation is less complete, more care is needed. The diet must be regulated, the bowels kept open, and the general health attended to. A weekly dose of blue pill or calomel, and digitalis once a week or more often over a long period of time, will often maintain and even increase the degree of compensation.

In every case it is essential to recognise the cause and differentiate the type of cardiac disease, and clearly to understand its probable course and prognosis. Our methods of treatment do not differ markedly from those of the past, but I think we may claim that they approximate more accurately to the needs of the particular patient, that we appreciate more clearly the necessity for prolonged rest and great care during convalescence, and that we do not hold such gloomy views as to the prognosis in heart-disease.

November 3rd, 1913.

THE PREVENTION OF MOUTH-BREATHING.

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THE ill-effects produced by nasal obstruction and mouth-breathing are so significant that the establishment of normal respiration is regarded as of the utmost importance. Nasal obstruction has received much consideration, whilst until recently little attention was paid to the direct effects of mouth-breathing. The pathological conditions which become established as a result of mouth-breathing are of the greatest importance. Impaired development of the bones of the face can be traced both to the obstruction of the nose and to the abnormal function of the mouth. The maxillary bones are those chiefly affected; they present several types of deformity. The two chief ones consist of non-development, where the teeth are found to erupt into positions corresponding to those found in the bones prior to eruption of the teeth, and a second type where marked deformity has resulted in protrusion of the anterior teeth.

A condition directly attributable to mouth-breathing is backward displacement (or lack of development) of the mandible. When the mouth is constantly open the muscles which support the mandible and the structures attached to it below are at a disadvantage. The weight of the pharynx and larynx constantly tend to displace the mandible in a downward and backward direction, and it is conceivable that this backward displacement may cause some impairment of respiration. Obstruction to respiration in this manner can be seen in patients under an anæsthetic, and with the mouth open, the freer respiration which results from pulling the mandible forward is well known. Improvement in general health has followed advancement of the mandible by means of dental appliances; the freer respiration seemed to be the only explanation. That a child suffering from nasal obstruction is usually less robust than a nasal breather is recognised although the explanations given are rather vague. Imperfect aëration of the blood is said to occur, although apart from mechanical obstruction as suggested above there does not appear to be any reason why respiration through the mouth should not be as free as through the nose.

The mouth is affected by the absorption of moisture by the air, which should normally be taken up during its passage through the nose as was pointed out by Greville MacDonald.

The malformations of the bones with irregular arrangement of the teeth, which are often imperfectly erupted, are very important as they predispose to local affections. The most serious pathological changes take place in the supporting structures of the teeth. In children gingivitis in the front part of the mouth is constant in mouth-breathers, and if uncorrected the condition of pyorrhœa alveolaris is certain to supervene.

In children gingivitis affects the gums of the incisor teeth both of the maxillæ and mandible; the gums are congested and swollen so that food particles are easily retained. Caries of the teeth is frequently seen; the gums are dry from the air passing over them and the absence of the normal bathing with saliva. In adults the loss of the teeth from pyorrhœa alveolaris is very commonly seen and may occur in quite early life: in a girl of seventeen the destruction of tissue was so extensive that the central maxillary incisors were so loose that they might have been removed with the finger and thumb. The tissues in the front part of the mouth are alone involved in the early stages, although infection of all the teeth follows if treatment is not undertaken. The recognition of mouth-breathing in adults may be overlooked in the less noticeable cases; the persistence of the habit at night, although controlled during the day, is only too common.

The recognition of pyorrhœa alveolaris in the front of the mouth should lead to a most careful inquiry as to whether the patient is a mouth-breather. Pyorrhœa alveolaris is detected by the slight swelling and blueness of the small papilla of gum which is situated between the teeth. This papilla presents a more rounded appearance and can be separated from the neighbouring tooth with a fine instrument. As the condition progresses, the papilla, although swollen, does not reach quite so high up the neck of the tooth as it should do, and a line is seen forming the base of a triangle which comprises the papilla. The change is due to loss of the summit of the piece of bone situated between

the teeth; the papilla of gum sinks down, and the outer bony edge produces the line described above by the gum folding over it.

Symptoms indicating mouth-breathing are often typical. A dry mouth or throat upon waking in the morning with an unpleasant taste are usual. The patient may wake in the night or the morning with a cough, which may be troublesome in the day, and nasal catarrh is generally present. The more severe cases have symptoms which are obvious and secondary conditions may be present.

The procedure adopted for the establishment of nasal respiration has consisted of operative measures followed by breathing exercises and voluntary

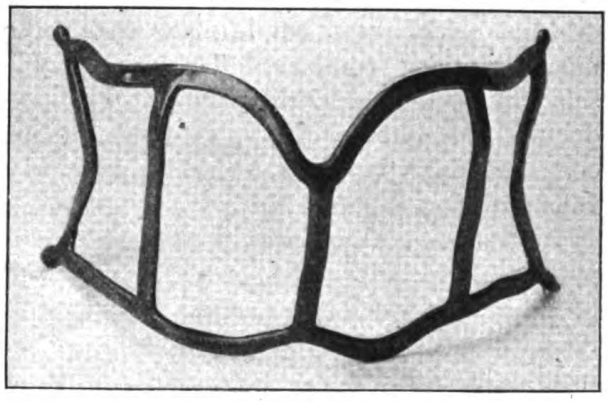

FIG. 1.*

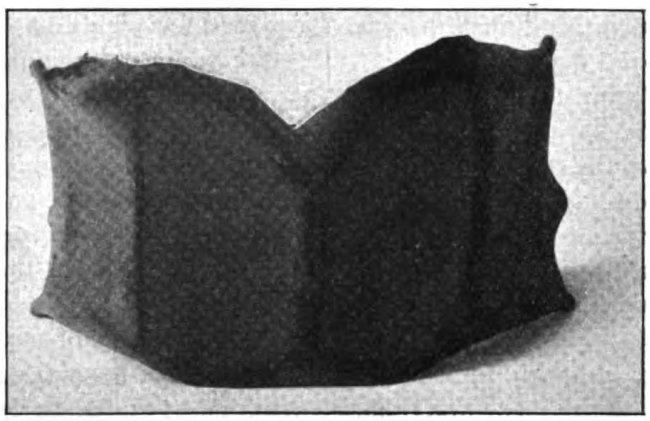

FIG. 2.

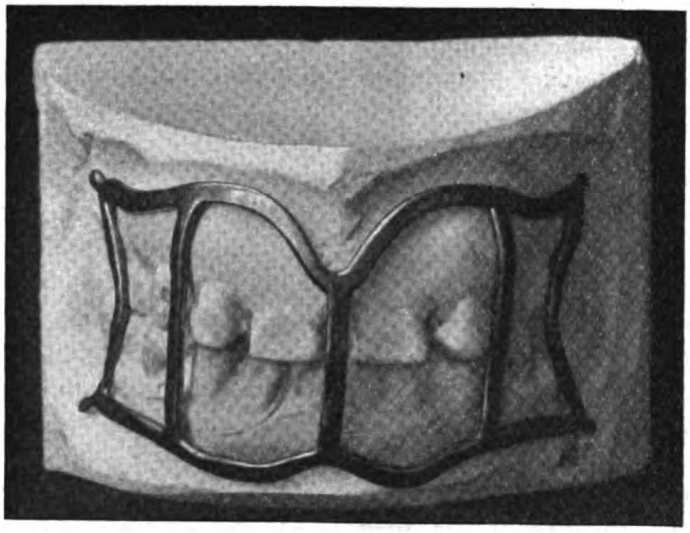

FIG. 3.

* Illustrations by courtesy of the Royal Society of Medicine.

efforts on the part of the patient. This will suffice in a number of cases, but in many patients the mouth-breathing continues. The nose may be sufficiently free for nasal respiration, but failure is due to the persistence of a habit which, although controlled during the day, recurs at night when voluntary effort is impossible.

The serious consequences, particularly cases of pyorrhœa alveolaris, which ensued upon mouth-breathing, and which it was useless to treat whilst the habit persisted, led the writer to endeavour to remove the cause.

In so many cases the common report by the surgeon that the nose was not obstructed and that the patient was now breathing through the nose led the writer to believe that many of these patients were mouth-breathers at night, and that this was the primary cause of the local dental condition.

After attempting bandaging the jaws to keep them closed or putting a bandage across the mouth some success was achieved by the use of rubber strapping. Narrow strips were placed across the lips; a different position for the strapping was selected each night, but in spite of the use of spirit and grease to remove the strapping much discomfort was caused. By the use of strapping some patients were cured of the habit, although there was a distinct tendency to a recurrence. The best results were obtained by the joint use of strapping and a four-tailed bandage.

About twelve months ago the apparatus described below was devised and is now being extensively used, for the most part with great success. The apparatus consists of a wire frame (Fig. 1) over which sheet rubber (dental rubberdam) is stretched (Fig. 2); it is placed inside the lips and cheeks, resting upon the outer surfaces of the teeth and gums.

The frame is made to a model (Fig. 3), which is obtained by taking an impression of the outer surface of the teeth and gums. The wires are fitted and soldered together and then struck between a zinc cast and a lead die in order to secure an accurate fit. Gold wire is perhaps the best, but German silver, gilded, is quite serviceable. The apparatus should be sufficiently large that it is steady when in position, extending back to the second premolars. No danger exists from swallowing or displacement with an apparatus of this size, although curiously enough nearly every patient or parent needs assuring that there is no risk.

The apparatus is worn only at night except perhaps for a few hours in the day in order that the patient may become accustomed to it, or to determine that nasal respiration is possible. In small children the apparatus may be used in the day without any rubber to prevent thumb-sucking—a habit most conducive to mouth-breathing at night when the thumb is withheld.

When the apparatus is in position mouth-breathing is quite impossible, although many patients, by opening the mouth widely, can obtain air below it—a fact which seems to be of consolation to them. There is little difficulty in quickly removing the apparatus if it should be necessary. At first many patients unconsciously remove the apparatus whilst asleep and in such cases the use of a bandage also for a few nights may be necessary.

In cases of obstruction to nasal respiration operative measures should be undertaken. The apparatus can be regarded as a means of determining the necessity or not of an operation. In young children when the necessity of an operation for obstruction of the nasal passages is uncertain the nasal function may be so developed that the operation becomes unnecessary.

The prevention of mouth-breathing is often so beneficial that the patient sleeps better, the mouth being moist and the throat not irritated, and on waking the unpleasant taste may be prevented completely. Chronic nasal catarrh in these patients may be markedly improved and in some cases entirely removed. One of my patients, an adult, was able to take a full deep breath through the nose for the first time in her recollection.

The length of time the apparatus must be worn varies. It should be continued until normal respiration is established. The process of discarding the apparatus consists of omission for one might as a test, and then to progress with omissions on one night in two, then one in three and so on. When patients have become thoroughly accustomed to the apparatus there is little difficulty in persuading them to continue with it; their comfort at night is often increased.

November 3rd, 1913.

THE DIAGNOSIS OF URETERO-VAGINAL AND VESICO-VAGINAL FISTULÆ.*

By A. H. BURGESS, M.Sc., F.R.C.S.,
Hon. Surgeon to the Manchester Royal Infirmary.

GENTLEMEN,—I wish to-day to speak to you of a type of case which, while it interests us chiefly from a diagnostic point of view, is one capable of causing very considerable distress to the unfortunate patient, and no small sense of disappointment to the surgeon concerned where, as usually is the case, it is the result of an accident during the performance of a surgical operation—I refer to urinary vaginal fistula. Of the three types of this condition—urethro-, vesico- and uretero-vaginal—the first presents little difficulty in diagnosis, inasmuch as urine escapes from the vagina only during the act of micturition, and on this account also it is a source of much less discomfort to the patient. It is to the diagnosis of vesico-vaginal and uretero vaginal fistulæ that I especially wish to refer. Moreover, in most cases of vesico-vaginal fistula, the aperture of communication is fairly large, and can be readily seen with the aid of a vaginal speculum, while its connection with the bladder is easy to demonstrate, as by the passage through it of a probe, which can be made to touch a metal sound introduced into the bladder *per urethram*, or by the well-known device of filling the bladder with an opaque or coloured fluid—such as sterilised milk—and observing its escape at the fistulous orifice. Where, however, the fistula is extremely small and runs a sinuous course, its vesical origin is much less easy to demonstrate—it may be impossible to pass even the finest probe along it, and milk may not be seen escaping during even a fairly long period of observation. And even if the fistula be definitely of ureteric origin, it is usually quite impossible to state either from the patient's symptoms or from an examination of the fistulous orifice itself whether the ureter concerned is that of the right or the left side—a question of vital importance when we consider that in many cases the only cure for the condition is removal of the corresponding kidney. To excise the wrong kidney in attempting to cure a condition which is itself due to an accident on the part of the surgeon would be an unspeakable calamity, and one very difficult to explain away subsequently to the patient or her friends. The question is further complicated by the fact that uretero-vaginal and vesico-vaginal fistula may co-exist, both resulting from the injury inflicted during the operation, and under such circumstances there may be only a single fistulous orifice in the roof of the vagina, the connection of which with both ureter and bladder being then far from obvious. Enough has been said to impress upon you the necessity in all these cases of carrying out a careful routine examination such as will enable you to state with absolute certainty whether the fistula is of vesical or ureteric origin or both, and if ureteric, which ureter is the one concerned. Further, inasmuch as, in the latter event, nephrectomy may have to be seriously considered, the functional capacity of the unaffected kidney should be ascertained.

The usual history of these cases is that, following upon some gynæcological operation—perhaps most frequently vaginal hysterectomy, but also after abdominal hysterectomy—the patient, either at once or after an interval extending to a few days, where the ureter has been caught in a ligature, is found to be partially incontinent of urine, and an examination shows that urine is escaping more or less continuously from the vagina. In many such cases, no doubt, spontaneous closure of the fistula eventually occurs and the incontinence disappears, but in others the condition persists as a definitely established urinary fistula, and after a time it becomes obvious that surgical measures are requisite for its closure.

The cases forming the basis of these remarks have all been referred to me during the past five years in this condition of well-established fistula, at periods varying from four to twelve months after the operation from which they resulted, and in all of them a careful examination with the aid of a speculum and a good forehead light disclosed merely an operation scar at the roof of the vagina, with a small fistulous orifice from which urine escaped in a more or less continuous fine trickle, and along which a probe could be passed either only for a very short distance or not at all. The question for decision was whether the fistula was

* Clinical Lecture delivered at the Manchester Royal Infirmary.

connected with the bladder or the ureter, and if the latter, with which ureter.

The following is the routine examination which I carry out in such cases. As a first step and in order to render the urine easily visible through the cystoscope as it escapes from the ureteric meatus it is necessary to colour it, this being effected either by administering a two-grain pill of methylene-blue about two hours before the examination, or else injecting deeply into the buttocks thirty minutes before about 4 c.c. of a sterile 4 per cent. solution of indigo-carmine in normal saline; the latter is the more reliable method, but may prove rather painful. A cystoscopic examination is next made, and the physical appearances and relative activity of the two ureteric orifices noted. If one ureter has been completely divided the corresponding meatus will appear smaller, and no "shoots" of blue urine will be seen escaping from it. If, however, the ureter has been only partially divided, blue urine will escape at both orifices, though the "shoots" on the injured side would naturally tend to be less powerful, since some of the urine of this side escapes at the fistula. Next, an attempt is made to catheterise both ureters. This is easily carried out on the uninjured side, but on the injured the catheter will be arrested, usually after passing ½ to 1 in., where the injury was inflicted during a vaginal hysterectomy, or at a little higher level after an abdominal operation. If the division of the ureter has been incomplete, although some urine can still escape along it past the fistula and so reach the bladder, yet, owing to cicatricial contraction at the side of injury the catheter is arrested there. On the uninjured side the catheter may be left *in situ* for twenty-four hours, and the urine collected therefrom examined so as to ascertain the condition of the corresponding kidney. Of course, where no urine at all escapes into the bladder from the injured ureter the urine passed *per urethram* must all have come from the opposite kidney, and there is then no necessity to leave the ureter catheter *in situ*. The final step is to definitely exclude the presence of a concomitant vesico-vaginal fistula, and this is done by exposing the vaginal orifice of the fistula through a speculum, taking a long strip of dry sterile white gauze, packing one end of the strip firmly against the fistula, and loosely plugging the vagina with the rest of it. A catheter is passed, and sterilised milk introduced so as to fill the bladder, care being taken in withdrawing the catheter that none of the milk escapes on to the gauze plug. After two or three hours (if the patient can refrain from passing urine for so long) the strip of gauze is removed, and that end of it which lay against the fistula, and which will, therefore, have been coloured blue by the urine escaping therefrom, is carefully squeezed, and the expressed fluid examined microscopically for the characteristic fat-globules of milk. Their presence conclusively establishes the existence of a vesico-vaginal fistula, and their absence proves the fistula to be of entirely ureteric origin.

Case 1.—Female, æt. 38 years, who twelve months before had undergone the operation of vaginal hysterectomy for fibromyomata in a neighbouring town. On the third day after operation the urine was noticed escaping from the vagina, and a vesico-vaginal fistula was suspected. A catheter was introduced *per urethram* and retained for a week and to it a Sprengel's pump was attached. The doctor in attendance stated that during the whole of the time the pump was used no urine escaped by the vagina—an observation difficult to explain in view of subsequent findings. This was held to prove the vesical origin of the fistula and a plastic operation was undertaken for its closure, but the sutures failed to hold, and urine has escaped freely from the vagina ever since. On examination a tiny opening was found in some scar-tissue at the roof of the vagina, practically in the middle line, and through which a probe could not be passed for more than a quarter of an inch. Never at any time had there been any unilateral pain in loin or groin, or other symptoms to point to the side affected. The urine was, therefore, coloured by administering two grains of methylene-blue in pill form, and two hours later a cystoscopic examination showed urine to flow freely from the right ureteric meatus, while none escaped from the left. A catheter passed only half an inch along the left ureter, but easily as far as the kidney along the right, and was left *in situ*. The bladder was filled with milk, and the orifice in the roof of the vagina inspected for thirty minutes, but not a drop of milk was seen to escape, although blue urine did so in fair quantity. From the catheter in the right ureter twenty-four ounces of urine were collected in twenty-four hours, the percentage of urea being 2·2. Left lumbar nephrectomy was performed a few days later and

proved a difficult operation on account of extreme obesity, the kidney being found slightly hydronephrotic. This completely arrested the escape of urine *per vaginam*, and the patient is now (three years after nephrectomy) in excellent health.

CASE 2.—Female, æt. 31 years, who twelve months ago had the uterus and appendages removed by abdominal section for extensive inflammatory disease. Urine had escaped from the vagina ever since the operation. In this case cystoscopy after administration of methylene-blue showed that urine was escaping from both ureteric orifices, but the right orifice was definitely smaller than the left, and the "shoots" proceeding from it were small and wanting in force, contrasting markedly with the more frequent and powerful ones seen at the left side. The left ureter was easily catheterised, and the catheter being left *in situ* twenty-eight ounces of urine were collected in twenty-four hours with a urea percentage of 1·8. An attempt to catheterise the right ureter failed, the catheter being arrested two and a half inches from the ureteric meatus, the ureter at this point having evidently been partially but not completely divided. On applying the milk test with vaginal packing as described above no fat-globules were found on the gauze, and thus the possibility of an accompanying vesico-vaginal fistula was excluded. At a later date the right kidney was removed from the loin, and was found slightly hydronephrotic. This completely cured the fistula and the patient is now quite well.

CASE 3.—Female, æt. 62 years, in whom vaginal hysterectomy for carcinoma of the body of the uterus, performed three months previously, had been followed ever since by the vaginal escape of urine containing some pus. Cystoscopy after injection of indigo-carmine showed that no urine escaped from the right ureteric meatus (which was displaced definitely outwards), while the left meatus discharged normally. The catheter was arrested on the right side three fourths of an inch from the orifice, while it passed easily to the left kidney, and a sample of urine collected from it contained no pus. The milk and gauze test was negative. She had suffered occasional pain and tenderness in the right loin, and a slight rise of temperature on two occasions. Removal of the right kidney, which was in a state of early pyonephrosis, cured the fistula.

CASE 4.—Female, æt. 53 years, on whom Wertheim's operation for carcinoma of the cervix uteri performed four months previously was followed by urinary vaginal fistula. The operator states that the right ureter was considerably displaced outside the bifurcation of the common iliac artery by a mass of glands, and was accidentally divided. As the general condition of the patient contra-indicated any unnecessary extension of the operation, both ends of the divided ureter were firmly ligatured, with the idea of producing atrophy of the right kidney. Almost immediately after operation urine commenced to leak from the vagina, and has since continued to do so. Cystoscopy showed no escape of urine from the right ureteric orifice, and a catheter passed only one inch along it. The left ureter was readily catheterised. The milk and gauge test showed definite fat-globules to be present on the end of the gauze in contact with the vaginal aspect of the fistula. This test was performed on three separate occasions, and each time the result was definitely positive, proving the existence of a small vesico-vaginal fistula, in addition to the right uretero-vaginal fistula, through which latter the greater part of the urine escaping unnaturally passed. The case fell later into other hands, and I am informed that the removal of the right kidney diminished considerably but did not completely arrest the vaginal escape of urine.

CASE 5.—Female, æt. 57 years, who ten months before underwent abdominal hysterectomy for advanced carcinoma of the cervix. The left ureter was found to have become involved in the growth; the lower portion was deliberately excised, and the proximal end implanted into the bladder. Urinary vaginal fistula followed. Cystoscopy after administration of methylene-blue showed that no urine escaped from the left ureteric meatus, nor could any abnormal opening be recognised on the left side discharging blue urine. The right meatus functioned well, and the ureteral catheter was readily passed. The milk-and-gauze test, carried out on three occasions, gave a positive result, showing that the fistula was connected with the bladder as well as with the ureter, and that nephrectomy would not cure the condition, which persisted until death a few months later.

November 3rd, 1913.

THE LUMLEIAN LECTURES ON INTRA-THORACIC ANEURYSM. Delivered before the Royal College of Physicians, London. By F. DE HAVILLAND HALL, M.D., F.R.C.P.Lond.

The two prime factors in the production of aneurysm are degenerative change in the wall of the artery, and strain. There can be no doubt that among the most important conditions giving rise to aneurysm is the endarteritis of syphilitic origin. The lecturer has found the disease at as early an age as fifteen years. The average age at death is, for women, forty-three, and for men slightly higher. The ascending portion of the arch is mostly involved. Sir Richard Douglas Powell has said: "The essential phenomena of aneurysm are, firstly, those derived from the pressure of a tumour on surrounding parts; secondly, those indicative of that tumour being a vessel tumour or diverticulum from the main artery. If in attempting to make a diagnosis we reverse this logical order of reasoning and attach primary importance to the evidence of arterial disease and insufficient weight to the presence or absence of those of tumour, there are many clinical snares and pitfalls awaiting us." The possibilities of error in diagnosis are almost limitless, but fortunately X-ray method of examination has done great service in its detection. In regard to treatment nothing new is stated, though the lecturer is of opinion that Sir William Macewan's method of "needling" deserves more attention than it has received.

CLINICAL SURGICAL DIAGNOSIS FOR STUDENTS AND PRACTITIONERS. By F. DE QUERVAIN, Professor of Surgery and Director of Surgical Clinic, Basle University. Translated from the fourth Edition by J. SNOWMAN, M.D. London: John Bale, Sons & Danielsson, Ltd. Pp. 779. 510 Illustrations.

The author states that this book is the outcome of years of association with students as a teacher and with medical practitioners at consultations. It will appeal to the general practitioner because the methods of investigation are those which can easily be carried out by him. The scheme of the book is to start with the symptoms which cause the patient to seek medical advice, and after appropriate questions, to narrow down the problem until a diagnosis is arrived at positively or by the process of gradual exclusion. This plan will commend itself in preference to the converse one which would deduce symptoms from a tentative or established diagnosis. The ripe experience of this teacher is reflected in an attractive book. Its perusal for reference or otherwise can only result in profit to the reader. The illustrations are the author's own, and are works of art. When faced with the question of differential diagnosis a reference to the text and corresponding picture will in the majority of cases solve the problem. The book is most readable and well got up.

TREATMENT AFTER OPERATION. By WILLIAM TURNER, M.S., F.R.C.S., Lecturer on Clinical Surgery, London School of Clinical Medicine, and E. ROCK CARLING, B.S., F.R.C.S., Surgeon, Westminster Hospital. University of London Press. Pp. 247. 10*s.* 6*d.* net.

On perusing this book one slowly realises the risks and dangers, even when those of the operation itself have been safely negotiated. The work has been produced with the object of indicating the possible complications and sequelæ of operations, and how best these may be obviated or dealt with should they unfortunately supervene. The range is of necessity wide, covering as it does major and minor surgery. The authors have pronounced opinions on the efficacy of continuous proctoclysis, but hold that good results are obtained only by those who practise it with scrupulous attention to details. It is held that, with an opening for draining in the abdominal wall, when proctoclysis is proceeding at the rate of two pints in three hours, or rather less, the normal current of absorption from the peritoneum into the circulation is reversed, and a profuse external discharge produced which constitutes the only efficient method of flushing the peritoneal cavity, the supreme advantage being that it proceeds from within outwards. There are interesting comments on the after-treatment of hernia and intestinal obstruction. The last chapter is on general considerations in treatment after eye operation. On account of the rarity of such cases attended by the general practitioner this chapter will be specially welcome. There are nearly a hundred plates and diagrams in the book.

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LECTURES
ON
THE DIFFERENTIAL DIAGNOSIS OF CHRONIC GASTRIC DISORDERS.*

By ROBERT HUTCHISON, M.D., F.R.C.P.,
Physician to the London Hospital, and Physician (with charge of Out-patients) to the Hospital for Sick Children, Great Ormond Street.

LECTURE I.

THE GENERAL EXAMINATION OF THE PATIENT.

GENTLEMEN,—In considering the differential diagnosis of chronic gastric diseases, the first thing we have to determine is, are the symptoms of which the patient complains due to disease of the digestive system at all? I say that because, as we shall see, there are very many diseases entirely outside the digestive system whose symptoms are such as to lead the patient—and sometimes also his doctor—to suppose that the disease is in the stomach or the intestine, when in reality those organs are perfectly healthy.

I shall consider with you therefore first, to-day, the general examination of a patient supposed to be suffering from a disease of the digestive system. In the next lecture we will consider those conditions which tend to simulate gastric disorders. In the third lecture we will consider the differential diagnosis of the organic diseases of the stomach; and the fourth will be devoted to the differential diagnosis of the functional disorders of that organ.

THE ANAMNESIS.

The general investigation of the case falls naturally into two divisions: (1) The interrogation

* Delivered at the London Hospital.

of the patient in order to elicit the history of his complaints; (2) the physical examination, or determination of the physical signs of disease present. As regards those two methods of examination, I have not the least hesitation in saying that the first is by far the more important, because in this respect disease of the abdominal organs differs greatly from, for instance, disease of the thoracic organs or of the nervous system. You can make a diagnosis of disease of the thoracic organs from physical signs alone, without knowing anything about the patient's history; and the same is true of organic disease of the nervous system; you are guided almost entirely by the results of your examination. In the case of the digestive system, however, that is not so; the history and the account which the patient gives you of his symptoms will usually help you to arrive at a diagnosis more than any amount of careful physical examination. It is important for you to get that fact firmly into your minds, because it follows, as a natural corollary, that no time spent in listening to the patient's account of his symptoms is thrown away. In the matter of taking the history you are apt to be handicapped by the loquacity of an intelligent patient, and by the stupidity of the less educated. Hence in this part of your examination you require great patience, and often a great deal of self-restraint. But it is well, even at the cost of considerable time, to allow the patient to describe the symptoms in his own words, you, on your part, avoiding, as far as possible, putting leading questions. Your part is to wait, and by a few simple questions to guide the information into the right channel. Through all the rigmarole which in many cases the patient will tell you, you must try to get clear the presence or absence of three important symptoms, viz. pain, vomiting and wasting. You must clearly ascertain whether the patient is the subject of real pain, whether vomiting is a prominent symptom, and whether he has lost weight materially. Those are the three most important points to elicit, and for the reason, as I have frequently pointed out to you in the wards and in the out-patient department, that if a patient exhibits these three symptoms or even one of them in any marked degree, it is almost certain that he is the subject of organic disease. And the therapeutic corollary which gives to the diagnosis its chief importance, is that organic disease of the digestive system is in most cases a surgical condition and calls for operative treatment. The chief *rôle* of the physician is to deal with functional disorders of those organs. I am strongly convinced of the truth and advantage of the more modern teaching in this respect, which we owe to the surgeons, that chronic organic disease of the stomach, ulceration, malignant disease, and so on, is best treated by surgical measures. On the other hand, the vast bulk of the cases which you will meet with are functional; and it is your business to eliminate the organic from the functional, so that you may know what kind of treatment to adopt. I scarcely know whether it is more disastrous to hand over a functional case to a surgeon, or to keep an organic case in the hands of a physician; both are certainly mistakes in most instances. So you see the importance of ascertaining what kind of case it is early, and this you do mainly by eliciting the symptoms.

I now want to refer in more detail to those three symptoms: pain, vomiting, and wasting.

Diagnostic Value of Pain.

In regard to *pain* your first difficulty will be to ascertain whether the patient is really suffering from what can be called genuine pain; because you will meet with many people who habitually speak in superlatives, and who use exaggerated language about everything so that they are apt to speak of every discomfort inside them as a "dreadful pain." It is your business to try and find out whether the patient's pain is really severe, or whether it is only a discomfort. In the case of more intelligent and educated people, it may be sufficient to ask them whether what they experience is really bad pain, or is it simply discomfort; but there are certain objective means of testing whether the patient has real pain or not. If the patient has been compelled by the pain to give up what he was doing at the time, and lie down, and especially if, in addition, he finds it desirable to apply a hot bottle to the stomach region, you may be fairly sure he has real pain, as opposed to mere discomfort—in other words, that it is caused by organic disease. Functional pains are not bad enough to cause the patient to cease what he is doing—whether it be work or play—and make him lie down and take other measures to get rid of the pain.

Having ascertained, as far as you can, that the pain is real, next direct your attention to its *site*. Remember that it is very difficult for patients to describe the exact site of pain in the abdomen. Anyone who has ever had a pretty bad abdominal pain knows that the pain has a way of spreading more or less all over the abdomen. Further, if the pain be not actually on at the moment of consultation, it is difficult for the patient to remember where it was situated, or where it began, because fortunately—or life would be impossible—our memory for pain is short. But although you cannot always get the patient to tell you exactly where the pain is, he will probably be able to tell you, in general terms, whether it is worse in the upper or lower part of the abdomen—that is, whether it is above or below the umbilicus. If the pain is above the umbilicus, it probably arises in the stomach, duodenum, kidney or gall-bladder. When the pain is below the umbilicus, it usually arises in the intestine or the appendix; or in the case of women, in the uterus or its appendages. Now, I do not deny that there are exceptions to these rules: I only say that, in general terms, they are true.

Assuming that you have roughly fixed the site of origin of the pain as either above or below the umbilicus, you have next to remember that some pains have a characteristic tendency to *spread*. Stomach pain, for instance, spreads to the back, and up between the scapulæ. In the case of duodenal ulcer the pain has much less tendency to spread. Gall-bladder pain spreads upwards into the chest and towards the right shoulder. Kidney pain begins, usually, in the back, below the ribs, and passes round the edge of the ribs and down into the groin. That is a very characteristic mode of spread. Appendix pain tends to radiate upwards towards the epigastrium. The pain of intestinal colic does not spread in any particular direction; it tends to remain round about the umbilicus. Hence by studying the direction of spread of the pain you may get valuable indications as to diagnosis.

The next thing to inquire about in reference to the pain is its *time relations*. The pain may be constant, in that it is there all day and every day. The pain of gastric carcinoma, for instance, tends to have that character. Again, the pain may be there every day, but not all day. The pain of gastric ulcer is like that; the pain is felt more or less every day; it comes on some time after a meal, and then passes away, coming on again after the next meal. In carcinoma the pain is there all the time. Then, again, pain may be intermittent—that is to say, it may be there for a number of days or weeks, and then there may be a longer or shorter period of complete immunity, followed by recurrence. That is very characteristic of the pain of duodenal ulcer—a pain which comes on at a stated interval after food, generally long afterwards, recurs for several days, possibly weeks, and then ceases quite suddenly, leaving the patient free from pain for a long period.

Or the pain may be periodic: it is not there every day, still less all day, nor is it intermittent in that it occurs in long spells, but there is one spasm, and then an interval of varying time, followed by another spasm. Pain of this kind is characteristic of biliary colic or renal colic; in fact all the colics tend to have that character.

The next thing you want to know about abdominal pain is its *relation to food*. The pain may have no relation to food at all. That always is a point which, *primâ facie*, is rather against the pain being a true digestive system pain. It makes it unlikely, but not impossible, because in all I am saying to-day there is no absolute rule; I am only describing what is usual. On the other hand, you must not hastily conclude that a pain which is definitely related to the taking of food necessarily has its seat in the stomach. Although gastric and intestinal pain almost invariably has some relation to the taking of food, the converse, that pains which have a definite relation to food are always gastric or intestinal, is not true. There are other organs in the abdomen, pain in which may be caused by taking food, and one of those organs is the gall-bladder. A patient with gall-stones not uncommonly gets pain a certain length of time after taking food, probably because the entry of food into the intestine sets up reflex contractions of the gall-bladder. There is such a definite relation of these pains to the taking of food that you are apt to be deceived and think the pains have their situation in the stomach itself. As a rule, however, as I have said, pain which is definitely related to taking food will have its site in the stomach or in the intestine. You find, for instance, that gastric ulcer pain is definitely related to the taking of food, the exact time at which it

occurs after a meal depending somewhat on the situation of the ulcer. In duodenal ulcer the pain is "late," it occurs two or three hours after a meal, and the taking of more food usually relieves it, hence it has been described as a "hunger-pain." But again, you must not suppose that hunger-pain is necessarily an indication of the existence of duodenal ulcer. We do not yet fully understand how hunger-pain is brought about, that is to say, how it is that the taking of food relieves the pain, but from the diagnostic point of view, although hunger-pain is most typical of duodenal ulcer, yet I have seen cases of appendix dyspepsia with typical hunger-pain; and it may occur in some cases of gastric ulcer, in which the ulcer is near the pylorus; I have found it also in gall-stones, and I have seen it even in a case of renal calculus, and in a case of carcinoma of the stomach. That shows the danger of attaching too much importance to any one symptom. Some surgeons speak as if hunger-pain always means duodenal ulcer, but that is certainly not true. But it still is true that the most typical cases of hunger-pain are those associated with duodenal ulcer.

Not only has the relation of the pain to meals to be considered, but also its relation to the kind of food taken. You will find, for instance, that in so-called gastralgia, which is apt to be mistaken for gastric ulcer, the pain is brought on by all foods, even by liquids. In true gastric ulcer, on the other hand, solids cause more pain than liquids. In cases of gastritis, again, the kind of food the patient takes affects the pain very greatly. On the other hand, there are many cases, such as those of duodenal ulcer, where the kind of food taken by the patient makes very little difference.

Next, it is necessary to consider the *relation of the pain to position or movement.* The fact of pain being relieved by the patient lying down has no diagnostic significance whatever, for nearly all abdominal pains are relieved by lying down. On the other hand, if the patient tells you that his pain is definitely brought on or made worse by more or less violent movement, such as shaking about, that is rather indicative, on the whole, of such things as renal calculus, or gall-stones, or a movable kidney. Mere shaking or jolting about does not tend to affect much the pain of duodenal or gastric ulcer. Pain which is much affected by the position of the patient is rather indicative of the presence of adhesions. I mean that supposing there is much more pain when he lies on one side of the body than when he lies on the other side, or if there is pain on raising the arms above the head, or, in a word, when he assumes any particular position, that is rather indicative of adhesions as the cause of the pain.

Lastly, you should always ask whether the pain ever *wakes the patient up at night.* There are some pains which tend to come on in the night, while there are others which practically never do. The pain of duodenal ulcer and of gall-stones often tends to waken the patient up. On the other hand, pain caused by a movable kidney almost never comes on when the patient is lying down.

Diagnostic Value of Vomiting.

I pass now to a study of the next symptom, which is much simpler, and that is the question of *vomiting.* There is no real difficulty in ascertaining whether a patient has been sick or not; the only difficulty is one which arises from the ambiguous use of terms. There are some patients who understand by "sickness," not vomiting, but nausea. It is well, therefore, to ask, not whether the patient has been "sick," but whether he has actually vomited or brought up food. If vomiting is a frequent and prominent symptom in the patient's history, the probability is that he is the subject of organic disease, because experience shows that functional affections of the stomach do not tend to cause vomiting. I want you to get that firmly into your minds, because it will save you from commiting many errors. On the other hand, I want to put this warning before you. There is a group of nervous affections which closely simulate dyspepsia, in which vomiting is the most prominent symptom, or at all events, a prominent symptom. Of these affections I will mention three. Migraine is one, so-called nervous vomiting is another, and the third is the gastric crises of tabes. You may say that nobody would ever mistake these conditions for gastric disease, but I can tell you that they do. I have known gastro-enterostomy performed for every one of them. So the danger of mistake is a very real one.

In addition to ascertaining the fact of vomiting, you may get some information from the *kind of vomiting*, and the circumstances under which it occurs. Take one or two illustrations. The vomiting

of mucus, particularly in the morning, points to gastritis, especially, perhaps, alcoholic gastritis. The vomiting of large quantities of frothy material points to organic dilatation of the stomach. The vomiting of food, more or less digested, some time after it has been eaten, and relieving any pain which may have been present, points rather to gastric ulcer. On the other hand, the vomiting of food fairly soon after it is eaten and comparatively little changed by digestion points to carcinoma, and so on.

Diagnostic Value of Wasting.

The third symptom which we have to consider is wasting. Here again you are apt to be deceived by your patient. Always get from him, if you can, definite information as to how much in pounds he has lost in weight in a given space of time. But always remember that there are patients with functional disorders who waste because of over-strict dieting to cure the disorder, and that may deceive you. Putting aside the possibility that it may be due to over-strict dieting, marked wasting points, like severe pain, to organic disease.

Other Abnormal Sensations.

Having ascertained these three cardinal important symptoms, you may now inquire as to some other abnormal sensations of the patient which are of less importance, and which point to functional rather than organic disease. A sense of "fulness" in the epigastrium, for instance, is usually an indication of functional disorder, especially of a want of tone in the stomach. A feeling of "sinking" or "emptiness" in the epigastrium, again, is a thing of which patients often complain, especially if gastroptosis is present.

You should also inquire about flatulence, and whether it comes on only after meals, which is genuine flatulence, or whether it is present when the stomach is empty, in which case it is generally due to swallowed air—so-called aërophagia or nervous eructation, a condition I shall speak of more fully later on.

The state of the appetite is also of diagnostic importance. Impaired appetite usually means diminished gastric secretion. It is, therefore, usually met with in cases of gastritis or gastric carcinoma. Usually when the hydrochloric acid is diminished there is a special loss of appetite for meat; this is a small point, but helpful sometimes in the diagnosis of gastric carcinoma. But where the appetite is increased, or at least maintained, you are probably dealing with a condition associated with an ample secretion of gastric juice. Gastric and duodenal ulcer are such conditions.

Lastly, you will inquire about the state of the bowels. With regard to constipation, I would say that it is a symptom which is so frequent as to be of no diagnostic value; diarrhœa belongs to another category altogether, but its diagnostic significance is too big a subject to enter upon now; later on I may deal with it fully.

The Physical Examination.

You may now pass to the second part of the investigation of the case, namely the physical examination.

I would emphasise, at the very outset, in regard to physical examination, that you must not be content with confining your examination to the abdomen, for, as we shall see in another lecture, in many cases simulating abdominal disease the real source of the mischief is somewhere remote, namely, in the chest, or in the general nervous system, or the urinary apparatus, or in the blood, and not in the abdomen at all.

You will note particularly in the general appearance of the patient whether there is anything to confirm your impression that the case is, or is not, one of organic disease. Does the patient, for instance, look wasted or cachectic? Cachexia points to organic mischief.

In regard to the examination of the abdomen itself, I shall only direct your attention to some special points, as you are already familiar with the general methods employed.

First, with regard to the *general appearance of the abdomen*. It is important to note the general shape, because there are abdomens which seem to confer on their happy possessors an almost complete immunity against functional dyspepsia; whilst there are others the possession of which makes it almost impossible to escape functional dyspepsia of one sort or another. The healthy kind of abdomen, that which does not render you liable to dyspepsia, is that having a wide subcostal angle. You will not often find a patient with an abdomen of that sort who is the subject of functional indigestion. If you feel inclined to diagnose functional

disease in an abdomen which has this wide subcostal angle, I advise you to think twice before you do so. The kind and shape of abdomen which renders the patient liable—for reasons I cannot give you now, and which, indeed, are not properly understood—to suffer from functional disorder, is a narrow abdomen with a sharp subcostal angle, that is to say, one in which the abdominal organs are rather crowded together in the upper part.

Again, you will note local fulness in the abdomen. An abdomen which is very hollow in the upper part, and very full below the umbilicus, particularly when the patient is standing up, should lead you to suspect gastroptosis, that is, a dropping down of the stomach. On the other hand, an abdomen which is very full above the umbilicus and comparatively empty below it, will make you think of the existence of a dilated stomach. An abdomen which is full round its periphery makes you think of distension of the colon, whatever it may be due to. An abdomen which is full in the centre, and comparatively empty in the flanks, makes you think of distension of the small intestine, very often with obstruction in the region of the cæcum. You may also be able to see peristalsis. When you are able to observe the peristaltic waves, they are always worth careful study. I would only put this general warning before you, however, with regard to peristaltic waves, that you should not attach much importance to them in people who have extremely thin abdominal walls, and where there is a wide gap between the recti muscles. The kind of case I mean is found particularly among elderly married women who have borne many children. In such patients, intestinal peristalsis may be easily visible even when it is normal, and I think in them also the normal movements of the stomach can sometimes be observed. But in the others, who are the vast majority, visible peristalsis should arouse a suspicion of the presence of obstruction. In the stomach you will find peristalsis passing from left to right, indicating always obstruction of the pylorus. In the transverse colon, peristalsis passing from right to left indicates obstruction about the sigmoid. In the small intestine you will get little superimposed coils standing out obliquely across the abdomen. Peristalsis in the small gut indicates, usually, some obstruction about the cæcum. Therefore a careful study of peristalsis, its direction and its situation, is of considerable diagnostic importance.

Next we come to *palpation*, which is the most important method of examining the abdomen. In your palpation you must proceed systematically, examining each region in turn—the left hypochondriac, epigastric, right hypochondriac and gall-bladder regions, right iliac fossa and kidney region receiving special attention. You will be on the look-out for the existence of tenderness, and—what goes more or less with tenderness—rigidity of the superimposed muscles, because this rigidity is simply an attempt on the part of the abdominal wall to shield the tender organs. I shall have something more to say about the significance of tenderness in these regions, but not just now. The other condition besides tenderness which you look out for is the presence of tumours. You should also try to elicit the presence of a splash in the epigastric or in the umbilical region. With regard to a splash, the tendency has been to attach too much importance to it, and if the abdominal wall be very thin, and especially if the patient has recently had a meal, the presence of a splash is not of much importance. What its real value in diagnosis is, I shall consider with you when I speak of dilatation of the stomach.

Lastly, you will proceed to percussion, *light percussion*, as it always should be when examining the abdomen, which gives you some information, though comparatively little, except in delimiting particular organs, such as the stomach. How you will use that method I shall refer to on another occasion.

Special Methods.

Those are the ordinary methods of examination. There are, however, two special methods of examination to which reference must be made: the use of test-meals, and the use of the X rays. I do not want you to get too much into the way of depending on these special methods of investigation. There are, for instance, very few cases which you can diagnose by means of test-meals alone. The test-meal furnishes valuable information, but it has to be taken into consideration with other symptoms and signs. Nor is the test-meal always capable of being applied; patients often object to it, and it is only in a minority of cases that you

need have recourse to it. The same remarks apply to examination by the X rays; it is a method which can only be carried out properly by an expert, and even in the hands of an expert it is at present full of fallacies. Experts, of course, will not accept that statement, but I should say from my own experience that X-ray examination of the alimentary canal is just as likely to lead you into error as any other. None the less, in certain cases it is a method which should not be omitted, as it is the most convenient way of estimating the tone and position of the stomach, just as the test-meal is the only way of ascertaining with certainty the condition of the patient's secretion. You can, however, get at nearly all those things indirectly by a proper study of the case on other lines, and that study should always come first.

Finally, you must consider the question of diagnosis by exploratory operation. It is often a method which is perfectly justifiable; and in spite of what is said it may prove the only method available, because I wish to impress upon you this fact: that you may be as careful and experienced as you please, and yet you will certainly find, in a number of cases—far more indeed than you would think—that it is impossible to say with absolute certainty what is the state of things inside the abdomen. If you have carried out the methods I have put before you, you can practically always say whether the condition is functional or organic, but what exact form of organic disease is present no amount of experience or skill will always enable you to foretell with certainty. The longer I live and the more I see of abdominal disease the more I am certain of that. Therefore remember that in many cases the ultimate and exact diagnosis can be made only in the operating theatre. And if you have rightly decided that a patient has organic disease, you can safely leave the precise diagnosis to be made by the surgeon.

November 10th, 1913.

Gonorrhœa in a Male Infant.—A. Smith and C. S. McKee report the case of a boy, æt. 17 months, unable to urinate. A hot bath relieved the condition, but later circumcision had to be done. Four days later urethral discharge was noted and bacteriological examination demonstrated gonococci. The source of infection was doubtful.—*Brit. Journ. Child. Dis.*, vol. x, p. 477.

SOME DISTURBANCES OF THE SINGING VOICE.*

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(*Continued from p.* 486.)

Treatment.

Treatment may be educational, general medical or special medical.

Educational Treatment.

Professors of singing realise that the preservation of the voice is important as well as its development, and they are keenly alive to the risks attending violent emission of the notes, wrong production, faulty methods of breathing (physiological and elocutionary), exaggerated *coup de glotte*, breathiness in singing, forcing the chest register upwards, and mistaking the class or texture (*tessitura*) of voice. It is very tempting, however, to try to make the very utmost of a fine voice and to get very near the breaking strain if not beyond it.

Can we, without trespassing on educational preserves, help the vocalist to avoid the results of indiscretion and to rectify them, apart from treatment of local or general medical conditions?

We may certainly use our experience in taking the measure of the individual and realising what his general strength will stand. As regards respiration, we may, as medical men, make a closer examination of the respiratory movements than the professor of singing even in his most paternal moments would permit himself. We may thus detect even very gross violations of the rules of breathing in singing in the pupils of the best teachers. Is this always the fault of the teacher? A thousand times "no." The criticisms so freely uttered are often entirely unjustified. We have to blame the pupil, if we have the right to blame anybody. Constantly, on asking a pupil whether the teacher had not instructed him to breathe in such and such a way he will say "Yes." Our inclination is then to say, "Why on earth don't you do it?" The reply will be "I forgot," or "I did it once, but did not trouble to continue it." Nature is

* Lecture delivered at the Polyclinic.

somewhat sparing in her gifts, and a fine singing voice is not always accompanied by a high order of intelligence. One would almost say that a fine natural singer is better without too exalted an intelligence. He may stop to think too much, and singing to be thoroughly pleasing *should* be absolutely spontaneous.

At the risk of being considered a heretic I am opposed to the teaching of the anatomy and even of the physiology of the voice to the student of singing, as I am convinced that the less he thinks of the vocal organs the better. On the other hand, it is quite desirable that *teachers* of singing should have a practical knowledge of the structure and functions of the organs.

Again, in the course of our laryngoscopical examination we may find obvious and unquestionable large size of larynx or equally obvious small size to help in the classification of the voice in spite of what some authorities on the voice have said, though the laryngoscopic data do not by any means always help in this respect.

To get a general idea of the patient's voice for singing I take the middle c^1 tuning-fork and let him sing very softly the sound "*la la la*" up to g^2, the space above the stave on the music in the female voice, but an octave lower in the male. It is by this means generally easy to arrive at a judgment as to his tone, the position of the break, whether he pushes the chest voice too high, whether he opens his mouth (quite an unusual accomplishment), how he breathes, and even as to his general intelligence (as revealed sometimes by the number of times he has to be shown before he sings the series of notes prescribed).

General Treatment.

The *general physician* has often an important word to utter, and the final judgment often rests with him as to whether a given individual has the physical capacity to follow the career of a singer. My colleague Dr. Hector Mackenzie has more than once stepped in to prevent a student from continuing a course which he saw would end in physiological disaster, or to encourage one to go on with those vocal studies which in a moment of depression he was inclined to throw up. The strength of the heart is tested considerably by singing, and I am convinced that the heart muscle sometimes yields under the strain put upon it by the long respiratory efforts which the singer is called upon to make.

The early detection of incipient tuberculosis is a constant source of anxious care to those who look after the health of the aspirants to the singing art. I have often, therefore, made the student cough in my presence and had the expectoration examined without conveying to the subject any idea of my disquietude.

This brings me to one of the most trying features in our dealings with singers—their nervous constitution. The least suggestion of need for care is apt to put them in a state of panic and bring down an army of relations and friends bristling with inquiries and even reproaches. On the other hand, a word of reasonable encouragement may be interpreted as an instruction that no precautions are necessary, that the memory may be relieved of the burden of retaining the wise regulations which are so easy to forget.

Special Medical Treatment.

I shall not enter into any details as to the routine methods of treatment required for such obvious conditions as adenoids, enlarged tonsils, nasal catarrh, deflection of the nasal septum, swelling of the turbinated bodies, etc., of which you have heard enough within these walls.

There is, however, one form of disease which has been illustrated in a number of my cases and which seems to me to be of more significance than it has been credited with, namely *hypertrophy of the lingual tonsil.* This was well-marked in fifteen of the cases. In seven it was removed and in six of these great improvement was reported, there being no report as to the seventh. For some years I employed various forceps, curved snares and guillotines with varying success owing to the elusiveness of the tissue and the difficulty in guiding the instrument. The galvano-cautery failed in many respects and Sir Felix Semon has uttered a warning against its use in this region. Latterly I have used no other instrument except Murphy's lingual tonsil guillotine, which I here show you. Its movement in relation to the lingual tonsil, tongue and epiglottis can be followed with ease and accuracy under the laryngoscopic mirror.

This excess of the lingual tonsil will sometimes hamper the movements of the epiglottis, and a good deal of the mischief caused by it is the pro-

duction of disordered sensations probably through the glosso-pharyngeal nerve. In many cases the feeling of swelling is merely subjective; there are two points which will help to decide this, viz. if the throat feels more comfortable during the swallowing of food, and if when touching the back of the throat, the throat does not contract to the finger, as it normally should do.

During the production of a clear tone, especially in the higher ranges, it is necessary for the epiglottis to be erect so that the resonance pipe may be as straight and patent as possible. This point has been well established by the observations of Spiess.* Any redundancy of the lingual tonsil would interfere with its elevation and thereby lead to impairment of the quality of the tone.

I do not intend to touch upon the many details in regard to *breathing* for singers. I can only say that with Dr. Aikin† I think the practice of good singers has been universally "to get as much breath as possible by all the means in their power—that is, by a free use of the ribs and diaphragm as prompted by instinct," in spite of what masters have written.

Steadiness of respiratory blast is, however, of as much importance for the production of clearness of tone in singing as is steadiness of bowing in playing the violin. Lower costal breathing probably gives greater expansion and steadiness than any other type of breathing. Abdominal breathing is, I think, best adapted for the utterance of staccato notes. No doubt forcible contraction of the abdominal wall may be injurious to the female, but discreetly practised by healthy people it can be made a valuable aid in the production and preservation of the voice.

Physiology and pathology teach that the immobility of the upper part of the chest-wall in the contracted position favours the development of disease in the apex of the lung, so that Mandl's veto on clavicular breathing may lead to mischief if accepted *au pied de la lettre.*

Dr. Halls Dally‡ has shown uncontrovertibly that spinal movements take place during inspiration and expiration and we may be grateful for his careful studies. The increase of space produced is, however, of comparatively small account in relation to the general chest expansion.

Anything which will take the singer's attention away from his throat is valuable, and therefore, on this account, the cultivation of lower costal and diaphragmatic breathing may be encouraged. I believe that many of the most striking results have been due to this factor.

Singers' nodules (occurring not so much in singers as in school teachers, from irritation set up by the breathing in of dry powdered chalk from the blackboard) or a *tendency to nodulation* at the junction of the anterior and middle thirds of the vocal cords are caused by excessive pressure at those points of the vocal cords, and there is no doubt that Holbrook Curtis,* of New York, has arrived at the treatment of this condition better than anyone else. He found that by the production of a certain sound the vocal cords could be brought together at a different point—that is to say, the exact edges which naturally came together during the patient's *coup de glotte* were turned a little and a different surface came in contact. My impression is that it is a way of helping the patient to get quickly on to the thin register, and in the production of the thin register I am quite convinced that there is a thinning of the vocal cords, while during the process of getting thinner they become slightly devascularised. Holbrook Curtis's method roughly described is to make the patient sing the syllable "pmaw" on a high "head" note, and very often I have found a patient able to sing, after practising this exercise, a note he was unable to reach before.

It is scarcely possible to convey in writing the exact nature of this exercise, though it will be readily appreciated by anyone who is accustomed to deal with the voice. One important point is to make sure that the tone resounds vigorously in the mouth, which is ascertained by twitching the lower lip by means of the finger and letting it return to its tightened position when the tone is found to be interfered with, and renewed. A similar proceeding is described in the humming exercise (*Summ-Uebung*) by Moritz Schmidt.†

School teachers should use a wet sponge or towel instead of a dry duster for the blackboard.

Difficulty in singing before an audience requires sometimes a dose of bromide, sometimes a dose of nux vomica, general encouragement and an innocent spray of mentholised oil. I have in a difficult case sprayed adrenalin in an emergency, but this practice is distinctly to be avoided; I have followed it up with weak chloride of zinc.

I have referred to cases of *paresis of the internal tensors.* The final touch for bringing the edges of

* 'Krankheiten der Oberen Luftwege.' By Moritz Schmidt and Meyer, Berlin, 1909.

† 'The Voice: An Introduction to Practical Phonology,' p. 28. By W. A. Aikin, M.D. Longmans, Green & Co.

‡ 'Medical Press and Circular,' October 26th and November 2nd, 1910.

* 'Voice Building and Tone Placing,' p. 154. By H. Holbrook Curtis, New York.

† *Loc. cit.*, p. 679.

the cords together is effected by the internal thyro-arytenoid muscles of the vocal cords, and when these muscles are paretic an elliptical space is left between the vocal cords on phonation. This condition is usually accompanied by general constitutional feebleness, which is sometimes the result of overwork and underfeeding. Apart from rest and support, a good effect is often obtained by means of moderate doses of nux vomica or ignatia with strophanthus, also the application of the interrupted current to the sides of the larynx.

Some nasal trouble is often present, but the commonest is what we call simple chronic rhinitis characterised by an abnormal increase of secretion of mucus, sometimes in rather an inspissated form; immense relief has been given to a number of singers by the use of a snuffing nasal douche with a lotion consisting of 96 gr. of biborate of sodium, 48 gr. each of chloride of sodium and salicylate of soda, 72 minims of pure glycerine, and menthol water (made like camphor water), 6 oz. "A teaspoonful in about one ounce of warm water."

There is one drawback to watery solutions for the nose—that is that they are apt to cause turgescence of the turbinated bodies; this can be counteracted by the use of such an ointment as —anæsthesin, 20 gr.; vaseline, 2 dr.; solution of adrenalin hydrochlor. (1 per 1000), 20 drops; paroleine, 2 dr.

It may be said in general that if the condition complained of is hoarseness or huskiness, we must look to the condition of the vocal cords, especially whether they are inflamed or paretic. In the former case rest to the voice is of most importance, while in the latter exercise must be advised when all inflammatory conditions have subsided. Want of resonance is in general an indication for attention to the resonating cavities of the nose and naso-pharynx. Discomfort or pain points to inflammatory states of the pharynx and quite peculiarly to the lingual tonsil.

Weakness of the voice calls for attention to the breathing, and to any circumstances tending to induce debility of the muscular, circulatory or nervous system.

It is important that singing aloud should be practised only for short periods daily. Much time may be beneficially employed in silent study of various kinds.

Diet and regimen are important factors, but I cannot help thinking that the meticulous details into which some writers enter in regard to these matters in the case of singers are apt to defeat their own object. Too anxious care in striving for health of body is apt to induce an unhealthy state of mind, such as is incompatible with the practice of singing as an art and still more as a profession. Sensibility is essential, but sense no less so.

November 10th, 1913.

CARCINOMA RECTI.

A Clinical Lecture.

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CARCINOMA of the pelvic colon and rectum is a condition of considerable importance to both the surgeon and physician, not only on account of its comparatively frequent occurrence, but because in a number of cases it may be overlooked or give rise to a mistaken diagnosis. Carcinoma occurring in this region is generally quite correctly classified as a cause of chronic intestinal obstruction, but the physician in particular must bear in mind that carcinoma may occur in this region, and even attain considerable size, without presenting any symptoms of obstruction. The writer has under his care at present, for example, an elderly lady, who has an extensive carcinomatous affection of the lower rectum and anus, who has not suffered from constipation, but, on the contrary, consulted her doctor on account of the troublesome looseness of the bowels, with consequent scalding of the surrounding parts.

PATHOLOGY.

With the possible exception of the pyloric region the pelvic colon and rectum are the most common sites of intestinal carcinoma. Why this should be so will probably not be decided until something more definite is known with regard to the cause of carcinoma in general; but the curved S-shape of the pelvic colon, and its sacculation; the lateral curves, with corresponding internal valves of Houston, of the rectum; and possibly the small so-called valves of the anus, all tend to arrest the passage of fæcal matter, which, in this stage, particularly where there is constipation, has become firm and even hard, rendering abrasion of the mucous membrane of the bowel and infection with carcinoma more than possible. While the general tendency is to regard the intestinal contents as the source of infection, it is a question whether, in some cases at least, infection, like that of the lips and tongue, may not proceed from without. As illustrating the almost certainly infective nature of carcinoma,

together with the possibility of infection from without, an experience related by Sir William Macewen is worthy of record. He was asked to see an elderly lady who had been troubled for a considerable period with constipation. On examination she was found to be suffering from carcinoma recti so advanced as to render radical operation impossible. Soon thereafter he was consulted by a lady who was not a relative, but who lived in the adjoining small house and who often passed days with her suffering neighbour, and who was found to have an operable carcinoma recti; and he was then asked to see the maid-servant of the first of these elderly ladies, a girl, æt. 25 years, who had an early carcinoma recti. It was found that the elder lady had regularly used an enema syringe to relieve the constipation, and that this syringe had been successively used by the neighbour and by the maid.

It would certainly appear in this case that the syringe was the vehicle of infection of the younger woman and the maid, it having become infected when used by the elder woman. This instance, which is not an isolated one, should further act as a warning to all who have to treat, nurse, or live beside carcinomatous cases to regard them as infective, particularly where utensils likely to come near the affected area, bedding, etc., are concerned.

The age-incidence of carcinoma in this region affords no exception to the rule that the disease is essentially one of later life, but, while this is the rule, it must be remembered that exceptions are not wanting, and further, that when the disease does occur in early life it is generally very malignant. Thus the writer was consulted by a young man, æt. 22 years, on account of abdominal distension, which on examination was found to be due to a carcinoma recti, the diagnosis being confirmed by an exploratory laparotomy. The abdomen was full of fluid, while the whole serous coat of the intestine and parietal peritoneum was studded with small carcinomatous nodules, proceeding from the primary tumour, which had extensively involved the serous coat of the rectum. Cases of carcinoma recti occurring at the age of 12 or even less, while very rare, are not unknown.

Sex would appear to exert but little influence upon primary carcinoma recti, but carcinoma of the bowel secondary to that of the uterus is more common than similar infection from the prostate or bladder in the male.

Among predisposing causes, constipation may be suggested, but as it is often a symptom of the disease its ætiological value is difficult to determine. Alcoholism, by lowering the vitality of the tissues generally, and causing congestion of the liver with consequent impairment of the hæmorrhoidal circulation, may also have an effect. The writer has come across some cases of advanced carcinoma recti in middle-aged people who were heavy drinkers, but of course many cases are met with where there is no question of alcoholism. Syphilis would appear to lower the resistance of the tissues to carcinoma in a marked degree, but this applies to carcinoma in all regions.

In a large proportion of cases the carcinoma is situated in the upper portion of the rectum—too low down and deeply placed to be readily palpable from the abdomen, and rather high up to be easily detected by a finger introduced *per rectum* unless it be a long one. Less often the tumour may occur about the anal orifice, and in a smaller proportion of cases it may be situated above the rectum, in the pelvic colon, where it is much more likely to be detected by abdominal palpation.

It generally begins as an ulcer of the mucous and later submucous coats of the bowel, and is of the cylinder-celled type, the cells breaking through the basement membrane and gradually invading the outer coats of the bowel. While the mucous, submucous, and muscular coats are readily involved, the serous coat (where the carcinoma occurs in a portion of serous coated bowel) appears to be more resistant, the carcinoma frequently attaining considerable size internally before the peritoneal cavity becomes invaded. Once the peritoneal coat is penetrated, however, the carcinoma spreads with great rapidity all over the peritoneum. The carcinoma may occur in the form of an ulcer, plaque or actual tumour, and varies considerably in its mode of spreading. In the majority of cases it is of the *annular type*, extending in a complete circle round the bowel, which, as it contracts, narrows the lumen and gives rise very gradually to obstruction. As the lumen becomes constricted, an additional strain is thrown upon the bowel above the obstruction, which in consequence hypertrophies, and so for a time preserves the necessary balance. This hypertrophy is not confined to the bowel in the immediate neighbourhood of the tumour and, in long-standing cases may affect even a considerable

portion of the small intestine. It is remarkable how very narrow the lumen may become before actual symptoms of obstruction supervene, careful dieting and regulation of the bowels enabling the patient to maintain fair health and attend to business for a prolonged period, which may extend to years. Such cases, however, may have transitory attacks of acute obstruction from blocking of the narrowed lumen by a small solid portion of fæcal matter or undigested food.

As a rule, dilatation of the bowel also occurs at a comparatively early stage, and while this dilatation may occur just above the seat of stricture, it not infrequently occurs quite a distance from it, the cæcum being particularly affected. When the cæcum is thus affected the patient may complain of pain and tenderness over the appendix region, which, taken together with the history of constipation, may easily lead to an error in diagnosis. In the later stages, when hypertrophy is no longer capable of exerting a force sufficient to overcome the obstruction, dilatation becomes marked, and, the bowel becoming congested and very unhealthy, ulceration may occur, with perforation and involvement of the peritoneal cavity from escape of fæcal contents. Perhaps a more frequent cause of peritonitis is perforation of the bowel at a point close to the carcinomatous growth, which has penetrated the whole bowel-wall and then given way.

While the form just described is the most common, it must be remembered that carcinoma does not always assume the annular type, but may form a large *ulcerous plaque* involving a considerable area of the bowel-wall, and destroying its contractile power. In such cases there is no decrease of the lumen. On the contrary, the bowel-wall becomes rigid and possibly even dilated, and thus the obstructive symptoms, which are generally so typical of the previous type, are replaced, in the early stages at least, by undue looseness of the bowels, which, as in the case already mentioned, may amount to loss of control. In the later stages of this type, however, carcinomatous masses may project into and narrow the lumen, and thus lead ultimately to obstruction as in the other form.

While carcinoma recti, like other forms of carcinoma, tends to spread by the lymphatics, it does so remarkably slowly. In the case of the rectum at least this may to some extent be accounted for by the presence of lymphatic glands in the outer coat of the bowel itself, which would presumably check the spread to the lymphatics lying in front of the sacrum. It has also been suggested that, owing to their regularly dealing with offensive matter, the rectal lymphatics have acquired a power of selective absorption, and that they exercise their discrimination by rejecting cancer! In some cases, particularly of the non-annular variety, the carcinoma may spread widely in the wall of the bowel itself; and Mr. Rutherfurd, of Glasgow, states that he once had a case in which carcinoma plaques had spread, by extension in the bowel itself, to the cæcum from the rectum. Where the growth is situated in the rectum below the peritoneal reflection, extension to the surrounding tissues takes place comparatively readily once the bowel-wall has been penetrated, and in lesser cases may lead to considerable fixation and consequent difficulty in removal, while in more advanced cases it may result in involvement of the bladder, vesiculi seminales and prostate in the male, and the vagina in the female. Where the primary growth is situated low down in the rectum, local extension, with subsequent breaking down, may lead to openings into the gluteal region, which may inflame, swell, and ultimately give way, a carcinomatous fistula forming between the bowel and the skin surface, through which fæcal matter may escape. Where the growth occurs above the peritoneal reflection — about 3 in. from the anus—direct peritoneal involvement may occur, the peritoneal cavity becoming filled with serous fluid, while the peritoneum and serous coat of the bowel become studded with small carcinomatous nodules. This form of extension may sometimes occur from a comparatively small primary focus, and is a possible source of error in diagnosis.

Involvement of the liver generally occurs only in the later stages, when the disease is advanced and already recognised, but it has been known to occur in a case in which the rectal tumour was entirely overlooked. Carcinoma recti is not infrequently associated with hæmorrhoids, which may develop as a result of straining, or from pressure upon the superior hæmorrhoidal vein, by the tumour itself, or the glands situated around the vein in the external wall of the bowel, which have been involved by the carcinoma. The co-existence of the two diseases should always be borne in mind

as more than a possibility, as certain symptoms common to both are apt to be attributed to the obvious hæmorrhoids, and the hidden carcinoma thereby overlooked.

Symptoms.

As already indicated, while discussing the pathological characteristics, the symptoms vary with the type of carcinomatous growth. In the more common annular form, constipation, often existing over a period of years, and gradually becoming less amenable to treatment by purgatives, is characteristic, and in some cases this may be the only symptom during a long period. Occasionally the stools become flattened by pressure against the growth, or may come away in small pieces. Slime is frequently passed along with the fæcal matter, partly contributed by the discharge from the ulcerating carcinomatous surface, and partly by the unhealthy mucous membrane of the bowel, and blood may also be derived from the same sources. Thus a history of constipation, becoming very gradually worse, and the passage of blood and slime should suggest the possibility of carcinoma, particularly in an elderly person, and the presence of hæmorrhoids, which also cause discharge of blood and slime, should not deter one from making a careful rectal examination. Sometimes the chief concomitant of the constipation is distension, and while general abdominal distension would assist the making of a correct diagnosis, a localised distension of the cæcum with pain and tenderness over that region would easily lead to mistake. Thus the writer was asked by a very careful physician to see a woman who suffered from obstinate constipation, which did not yield to quite a series of purgatives which had been tried, and who complained of pain and tenderness over the appendix region. The case had been carefully studied from a medical standpoint, and a diagnosis of some appendix mischief, with consequent paresis of the cæcum, had been arrived at. A rectal examination, however, revealed a carcinomatous stricture some four inches from the anus.

In the non-annular or plaque type, on the other hand, constipation is generally absent, and the patient may rather consult a doctor on account of persistent diarrhœa, incontinence (particularly if the anal region be involved), and probably the passing of blood and slime.

In some cases particularly of this type the disease may have been long present without the patient being aware of it, the passing of blood being perhaps the first symptom to alarm the patient and suggest a visit to a doctor. Sometimes when a considerable tumour mass has formed, the patient may complain of a symptom frequently found in enlarged prostate—the feeling that the bowel is never properly emptied, a mass constantly remaining behind which cannot be dislodged, even with severe straining.

While absence of pain is often a noticeable feature of carcinoma recti, pain may occasionally be severe and cause much suffering. The writer has known such pain to persist even after palliative colotomy had been performed, the patient coming to him to have a radical operation performed in the hope that the pain would thereby be relieved. Carcinoma recti, likewise, by intra-pelvic pressure upon the obturator nerve or sciatic trunk, may give rise to pain referred to the knee, leg, or foot, and thereby cause error in diagnosis.

In many cases the almost entire absence of symptoms is a remarkable feature, which may cause the condition to be overlooked for a prolonged period, while in others, when symptoms do occur, they may be referred to some other condition. Thus an American gentleman was troubled for years by the condition of his liver, and had consulted many of the foremost physicians in America and Europe regarding it, who had supplied him with an equally numerous collection of prescriptions. Ultimately he visited the late Sir William Gairdner in Glasgow, and only on leaving remarked casually that he had occasionally passed a little blood when at stool. This led to his visiting a surgeon, who made a rectal examination, and the discovery of a primary carcinomatous focus there, the affection of the liver being really a secondary carcinomatous one.

While the obstruction caused by carcinoma is generally of a chronic type, it is sometimes of an acute character, and this is perhaps more likely to occur when the annular obstruction is in the pelvic colon. Here the fæcal matter is generally softer, and thus symptoms of obstruction may not arise until some large particle becomes lodged, when symptoms of acute obstruction speedily arise. A carefully administered enema may sometimes relieve such obstruction by dislodging the obstruction and perhaps facilitating its passage. When obstruction does arise, fæcal vomiting is not likely to be an

early symptom, owing to the obstruction being low down; but pain, referred to the region of the tumour, to the appendix region, and to the umbilicus, together with abdominal distension, often most marked over the cæcal region in the early stages, occurs. As the condition progresses the temperature tends to rise, while the pulse becomes fast and thready in the later stages. Where acute obstruction supervenes upon the chronic, abdominal distension may be very great, the abdomen sometimes assuming somewhat the appearance presented by a large ovarian cyst, but tympanitic at parts, while evident peristalsis may be present.

Diagnosis.

Diagnosis founded upon the symptoms described and confirmed by careful rectal examination is as a rule easy. Save in urgent cases the bowels should be well cleared out by purge and enema prior to making the rectal examination, and the patient should be asked to strain down, as this facilitates the passage of the sphincter by the finger, and brings the tumour down upon it. Having detected the tumour, which is generally hard and rough as compared with the normal mucous membrane, it is desirable to estimate as far as possible its size and mobility. The latter point, however, is not always easy to determine with accuracy. The pelvic colon should also be carefully palpated and the condition of the abdomen generally determined, while the liver must be examined very specially. Where from any reason there is difficulty in diagnosis it is generally easy to examine the rectum visually by placing the anæsthetised patient in an inverted position and introducing a speculum through the anus, which may be stretched if necessary. The rectum fills out with air and a very good view of its interior is thus obtained. Or the proctoscope may be introduced, the bowel carefully distended with air, and its interior then examined. In skilful hands the pelvic colon may thus be inspected from the interior. An injection of a bismuth compound may also give valuable information as to the presence of a stricture by taking one or more X-ray photographs during or after the injection. Unfortunately, however, the pelvic colon, which is the part concerning which information from such a method would be most useful, is difficult to skiagraph satisfactorily in many cases. Where the tumour is situated about the anus there is generally no difficulty in arriving at a diagnosis, but in case of doubt a small portion could be removed for microscopical examination.

The stricture most likely to be confounded with the carcinomatous one is that due to syphilis. As a rule it is situated near the anus, may present an almost sharp border if healed, in other cases an ulcer. In cases of doubt a Wassermann reaction would almost certainly remove any difficulty, but the possibility of carcinoma growing upon a syphilitic base must not be forgotten. While much work has been done, notably by v. Dungern and Ascoli, with a view to producing a test for carcinoma comparable to that of Wassermann for syphilis, the results so far obtained are not sufficiently reliable. Single tubercular ulcers about the lower end of the rectum generally occur in younger subjects who present other symptoms of tubercle, and may attain considerable size.

Prognosis.

Prognosis of carcinoma recti depends upon a number of factors, of which the age of the patient is one of importance. In young persons the prognosis, unless the condition be detected very early, is generally bad, if not hopeless, owing to the rapid course of the disease and comparatively early involvement of the general peritoneal cavity. In elderly persons, on the other hand, the growth is often very slow, so that, barring obstruction, a patient may live for a very considerable period—even years—without an operation. Where a palliative colotomy is done, the prognosis, in the case of an old person, is often quite good. Thus a patient who, when first seen, had an inoperable carcinoma recti, has been known to live for a period of seven years after the palliative operation had been performed.

The patient's general health, as regards the effects of alcohol and syphilis, kidney affections, etc., is also of importance.

The stage at which the disease is first seen has, of course, a very important bearing upon the prognosis. Thus in the case of a middle-aged person in fair health and with a fairly early carcinoma a radical cure can generally be effected, and this is often the case even when the patient is elderly and the carcinoma advanced. Where the primary tumour is inoperable much can be done by colotomy to prolong life and improve the patient's

general condition. Where the peritoneum is extensively involved—that is, well beyond the limits of the tumour—the outlook is hopeless and the course of the disease generally rapid.

Treatment.

Treatment in the present state of our knowledge is almost entirely confined to operation. Much is being done by the screening of X rays to permit of the passage only of such rays as affect the deeper tissues, and thus of employing larger currents than would otherwise be possible, and by the use of radium, thorium compounds, etc. (which might be introduced *per rectum*). But while several surgeons, and notably Kroenig, the gynæcologist, of Freiburg, can point to a series of apparent cures, professional opinion is not yet satisfied, more particularly as the writer's former teacher, Czerny, the Director of the Cancer Klinik in Heidelberg, who has done a vast amount of work in this direction, regards all such methods as valuable adjuncts, but not as methods of cure. Much is also being done with a view to finding materials which, when injected, will bring about destruction of the carcinoma. In Czerny's klinik many attempts have been made, particularly by injecting materials similar to those found in the tissues after treatment with X rays, such as cholin, to destroy the tumours, but so far without definite success.

With regard, then, to operative treatment, the writer favours the performance of a preliminary colotomy in the great majority of cases, and for various reasons. Where obstruction is threatening, or has already occurred, colotomy is the obvious treatment, yielding not only immediate relief, but, as already pointed out, often enabling the patient to live for a prolonged period and in an improved state of health. But even where there is no question of early obstruction a preliminary colotomy cuts the affected portion of bowel out of circuit and permits of its being regularly washed out and so kept clean. These points are of much importance to the patient, as under this treatment the tumour, no longer irritated by fæcal matter, generally shrinks and becomes much more distinctly circumscribed, rendering radical removal on a subsequent occasion much more easy, while the increased comfort and improved health of the patient are generally marked. For these reasons a palliative colotomy is well worth doing even in a case of inoperable carcinoma recti, and while some physicians profess repugnance to a colotomy for æsthetic reasons, the trouble involved in attending to a colotomy is negligible as compared with the relief, longer life and improvement to health obtained. As a preliminary to a radical operation there are the further advantages that the nourishment of the patient need be but little interfered with by the subsequent more severe operation; the part to be removed can be rendered comparatively clean; and, as already stated, the tumour has generally become much more clearly defined. In cases where the tumour is situated in the pelvic colon it can be excised through an abdominal incision as for colotomy and an anastomosis performed, the abdominal wound being closed after the bowel has been returned. Where the patient is in a very weak state it has sometimes been possible to pick out the portion of affected bowel, rapidly complete the colotomy operation, and subsequently, when the patient has recovered, remove the carcinomatous portion of the projecting loop. Without attempting to describe the details of the colotomy operation, attention might be drawn to one or two points. The upper limits of the carcinoma can often be accurately determined through the colotomy wound and the advisability of performing a subsequent radical operation gauged. In choosing the loop of bowel for the colotomy a portion as far up toward the splenic flexure as possible should be chosen, as thereby plenty of slack bowel is left for the radical operation and the risk of subsequent prolapse through the colotomy wound greatly diminished. The skin and other parts should not be brought together so as to press unduly upon the limbs of the loop, as otherwise immediate obstruction with great abdominal distension and possibly disastrous consequences may ensue. When there is no urgency it is well to defer the opening of the bowel for several days after performing the colotomy.

As soon as the patient is fit after this operation, knows how to attend to the part, and has had a colotomy belt fitted, he is advised to take several weeks holiday, so as to get his health into a thoroughly good condition. For removal of the tumour itself, the writer has found Kraske's operation all that can be desired. As a rule, by removal of the coccyx and possibly the last one or two segments of the sacrum, abundance of room is

secured. Where colotomy has already been performed some time previously, the location of the tumour and separation of the affected bowel from the surrounding tissues presents no special difficulties. Wherever possible—and when the tumour is in the common situation it is always possible—the last couple of inches of the rectum with the sphincters should be maintained intact. In order to get down sufficient bowel from above to effect an easy junction with the last inch or so, it is generally wise, unless the tumour be an extremely small one, to open into the peritoneal reflection to the rectum all round at an early stage, and before the bowel has been divided. Thereafter the bowel comes down readily, the mesentery of the pelvic colon coming into view as the bowel is brought down. If necessary, a portion of this mesentery can be ligatured and divided, and the bowel thus brought still further down. Then the peritoneal cavity having been carefully closed off, or, if necessary, packed, the bowel is divided fully an inch above and below the apparent limits of the growth, and an end-to-end anastomosis performed. The anastomosis does not require to be performed with the care necessary where the bowel is to functionate immediately afterwards, and the operation is thereby materially shortened. The skin wound is stitched and a short gauze drain left in. Immediately after the operation the patient can have saline or nutrient injections through the colotomy opening, and thus stimulation and feeding are greatly facilitated. After the patient has quite recovered he may have a holiday or return to business, and at some subsequent period he can have the colotomy undone and the abdominal wound closed. A patient so treated, then, has ultimately no colotomy wound to trouble him, and he has complete control of the action of his bowels, owing to the sphincters having been maintained intact. While in a sense a prolonged procedure, the advantages of this method are that no single stage is unnecessarily severe, nor, in the case of a business man, is he unable to attend to business during any very prolonged period. The writer has utilised this stage method of operating in some cases where the patients were heavy drinkers, their condition at no time being good. The fact that they made excellent recoveries says much for the method, as most surgeons will admit that a Kraske is a fairly severe operation.

Where the tumour is situated at the anus this method of treatment is not available, and where it is at all advanced, a radical operation involving removal of the sphincters is generally required. In such cases it is seldom necessary to perform a preliminary colotomy, and while the patient will subsequently generally suffer more inconvenience from removal of the sphincters, the operation is a less severe one.

In the case of a wide-spread carcinomatous affection involving pelvic colon and rectum, another method may be employed, which is preferred by some to the Kraske, even for circumscribed affections. A wound as for colotomy is made, the pelvic colon picked up and divided, and the upper end arranged as an artificial anus either through the existing wound or another. Then the mesentery of the pelvic colon is divided right down to the rectum, and the bowel, the divided end of which has been carefully closed, is dropped into the lower pelvis and covered by peritoneum, and the abdominal wound closed. Thereafter, on the same or preferably on a subsequent occasion, the lower end of the rectum is detached through an anal incision, and the whole portion of bowel removed. It is claimed for this method that there is less fear of peritoneal infection, but the writer has had no trouble with peritoneal infection with the Kraske method, while this method, while at least as severe as the Kraske, leaves the patient with a permanent colotomy. He, therefore, prefers the Kraske method in all cases where the carcinoma is not too widely diffused to make the method practicable.

In conclusion, the necessity of bearing the possibility of carcinoma recti in mind, even where the age or symptoms do not appear at first sight to render its presence probable, may be urged, careful palpation of the iliac region and no less careful rectal examination being made before it is excluded. A diagnosis of carcinoma having been made, its possible infectivity should be guarded against, and steps taken to promote the comfort of the patient by palliative or radical operation, while, if he knows that he is suffering from "cancer," he should be comforted by a knowledge of the comparatively good prognosis afforded by carcinoma recti in elderly persons.

November 10th, 1913.

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A FEW EXAMPLES OF GRAFTING.

By ALEX. MACLENNAN, M.B., C.M.,

Assistant Surgeon, Western Infirmary; Extra Honorary Surgeon, Royal Hospital for Sick Children; Honorary Consulting Surgeon, Stirling Royal Infirmary; Consulting Surgeon in Glasgow to the East Coast Railways.

SINCE the classic work of Macewen on bone grafting the interest in the subject of grafting has lately been greatly enhanced by the work, among others, of Carrel in America and Lexer in Germany. The former has carried out many remarkable experiments showing, at least, the possibilities; while the latter has successfully demonstrated what can actually be done in the way of restoration in the human subject. A review merely of the outstanding features of this branch of modern plastic surgery, while being intensely interesting, would be much too long and not at all suitable for an article dealing with the more clinical aspects of the matter. The present communication will refer to some cases occurring in the practice of the writer, which may be of general interest and which may serve as examples to anyone working in restricted circumstances and with limited opportunities.

(1) *Skin grafting.*—(*a*) In the 'Practitioner' for July, 1913, the author has described two methods of skin grafting which may successfully be adopted in various circumstances. "Tunnel" grafting has given most excellent results; and for those intending to try the plan that publication may be readily consulted. The underlying principle is the utilisation of the granulation tissue as the "protective" for the graft. Hence the grafts of skin are imbedded in cellular tissue under the granulation tissue layer. These grafts may be long or short, but should be narrow, so as to give a relatively large border for subsequent expansile growth.

Another plan is to place a graft under the granulation tissue shaped like a star fish, which may suitably be called "star" grafting. A piece of healthy skin is cut from the abdomen or inner aspect of the thigh in the shape of a star fish. The resulting wound is quickly closed by a suture approximating the free angles, and the gaping limbs of the star are closed by two or three interrupted sutures. The excised star is quickly transferred to warm saline. Tunnelling under the granulations is done from a centre radiating out like the spokes of a wheel. Five limbs of about 1½ in. in length suffice. The star-shaped piece of skin is then imbedded and the limbs located or not by strands of horse-hair lying along the tunnels. The localisation of the buried grafts by horse-hair has been described, in the article referred to, as "buoying."

The accompanying figure shows the shape of the star graft and the manner of closing in the space from which it has been taken (Fig. 1).

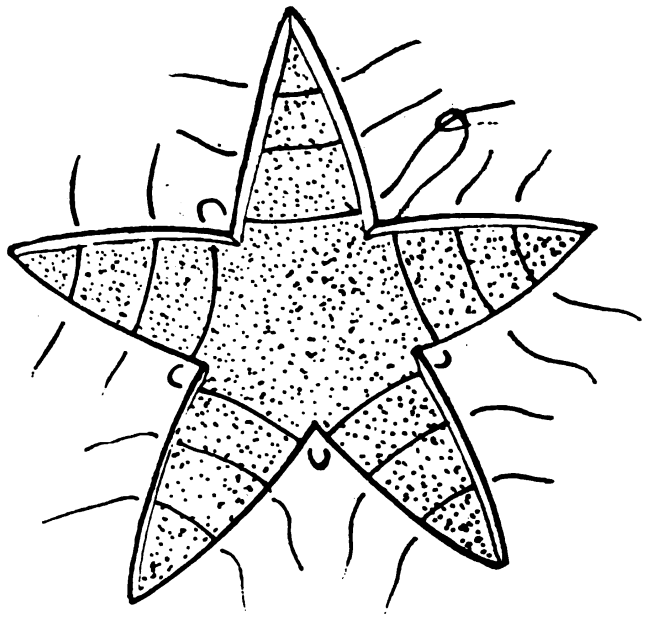


FIG. 1.—Gap in skin showing suture approximating all the angles, with two extra stitches to close the remaining gap.

Lately (1) some experimental work on the quickness of disappearance of imbedded grafts of skin has been published, and twenty-four hours have been stated to be the usual time for such pieces of skin to be incapable of further growth or life. The writer has ample material to disprove this limitation, but at the same time he admits the liability of imbedded detached fragments of skin to spontaneous disappearance as other absorbable foreign bodies. If, however, these grafts "hold," and are uncovered in ten days, few will be missing. Threads of horse-hair enable the surgeon to locate these grafts after they have have been completely covered in by granulations. If the skin pieces necrose they, of course, quickly liquefy and disappear. In the writer's opinion it is the presence of sepsis which determines the death of the graft.

(*b*) Grafting skin on to the face presents some difficulties, and often taxes the ingenuity of the operator to the utmost. When skin containing hair follicles is transferred from any part of the body to the face, hair usually grows most luxuriantly; hence the necessity for utilising hairless skin. The palms of the hands, soles of the feet and the penis are parts which provide hairless skin. The circumcised foreskin makes a most excellent graft.

Figures 2 and 3 show the condition of a nose before and after excision of the affected skin, with foreskin grafting.

(*c*) To restore a partially destroyed eyelid the following procedure was successfully carried out: A small piece of the margin of one ear was denuded of skin, the gap left being filled in with a small strip of foreskin. When this had become well attached the whole section of ear with cartilage was excised to the desired extent so as to reconstitute the missing part of the eyelid. Unfortunately the man has been loss sight of, and a photograph of the result has not been procured. The patient was entirely satisfied with the result.

(*d*) A small wedge may conveniently be cut out of the ear to fill in any slight deficiency in the nostril; as, for instance, in a case where the nostril involved in a wart has required to have a wedge excised. Fig. 4 shows the result of such excision with insertion of an ear-graft to restore the continuity of the nostril.

(*e*) In web fingers, as a modification of the writer's method (2), the circumcised foreskin, denuded of its so-called mucous membrane, makes what is subsequently found, after separation of the fingers, a most excellent web. The procedure is carried out as follows: The uniting tissue of the fingers at the level of the proximal ends of the first phalanges is stabbed through with a scalpel. The cut made on the dorsum is longer than that on the

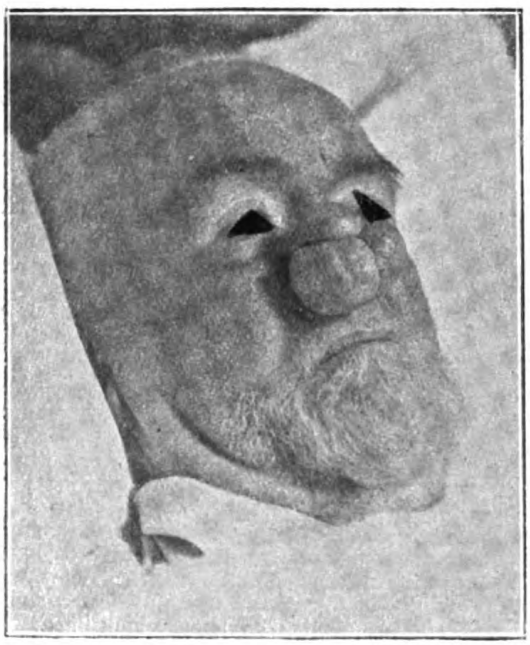


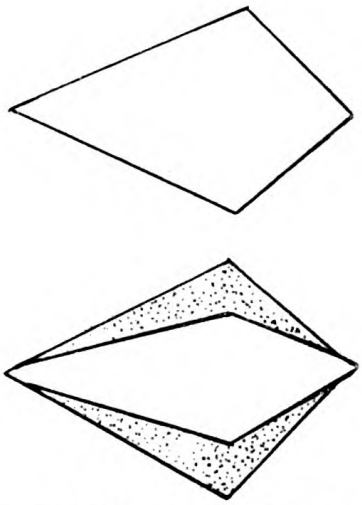
FIG. 2.—Lipoma nasi before operation.

FIG. 3.—Same case after excision down to cartilage with immediate application of circumcised foreskin, which was stitched in position.

palmar aspect, on account of the incision being enlarged in an upward direction. The object of this longer dorsal cut is obvious, if one looks at the normal web, which is developed only on the palmar aspect. Any fat which herniates through the incisions is snipped away. A slit digital artery perhaps may require ligature. The little tubular graft is then passed through the tunnel, narrow end towards the palm, and stitched in position. Before doing so, however, the graft has to be turned inside out. If there is a tendency for oozing to continue, a narrow strip of gauze should be threaded through the tunnel so as to exert slight pressure. Where more than one finger has to be separated, a piece of skin taken from anywhere convenient should be used instead. Figs. 5–9 show the way to shape the graft, etc. The result of these procedures is shown in Fig. 10.

FIG. 4 —Shows wedge removed with wart replaced by wedge from ear containing cartilage.

(*f*) Figs. 11–12 show a method of bridging over a large raw surface with its results. In this instance the ulcer was the result of a very large carbuncle in an elderly man. A plan, therefore, had to be adopted which would not entail the loss of any large amount of tissue in the event of failure. A long, wide flap was cut down the back from the edge of the ulcer through the entire thickness of the skin. At the base the skin all round was widely undermined to permit of an easy stretch without tension. The flap had its edge pared of ulcer-margin, and the scalp was likewise notched to fit the square end, which was stitched accurately to it. As the flap is pulled into position



FIGS. 5 AND 6.—Shape of skin to be used for tunnel graft in making new web for syndactylosed fingers.

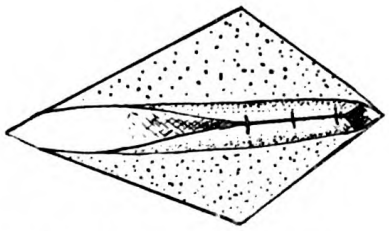


FIG. 7.—Skin after stitching so as to form a tube before final separation from bed.

FIG. 8.—Final appearance of graft after having angle at end cut off, ready for passing between fingers to form the new web.

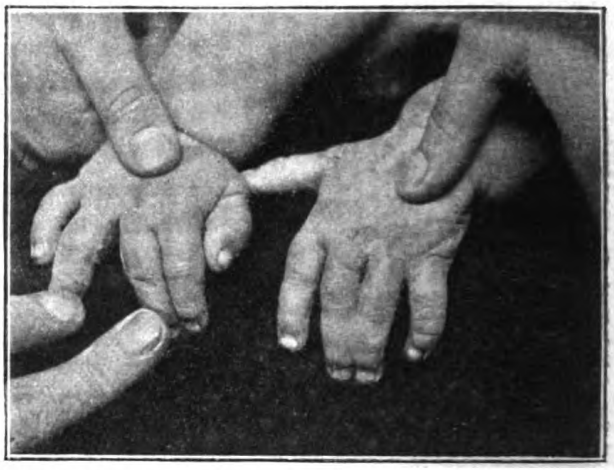


FIG. 10.—Case after operations completed in right hand. Fifth and fourth fingers of both hands separated by method described (2). Fourth and third of right hand grafted at web with circumcised foreskin, subsequently completed by splitting and stitching. The left hand shows a graft of skin taken from the abdomen, tunnelled through the united fourth and third fingers. The separation has since been completed with perfect result.

the skin at the sides tends to gape; to close this in, the sutures should be passed under the graft-flap to bring the outside edges into apposition with it, thus avoiding traction on the flap. On no account must the flap be stitched to the lateral margins, as this would interfere with the blood supply too seriously.

(2) *Nail grafting.*—The appearance and utility of the finger is much improved by the transplantation of a nail when this structure has been de-

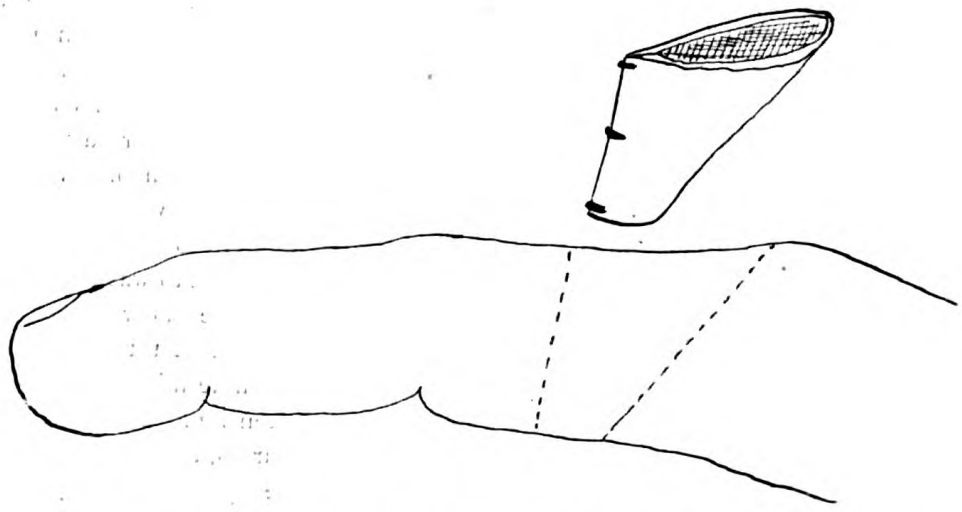


FIG. 9.—Shows shape of stab through web to fit stitched graft in position ready to be passed through.

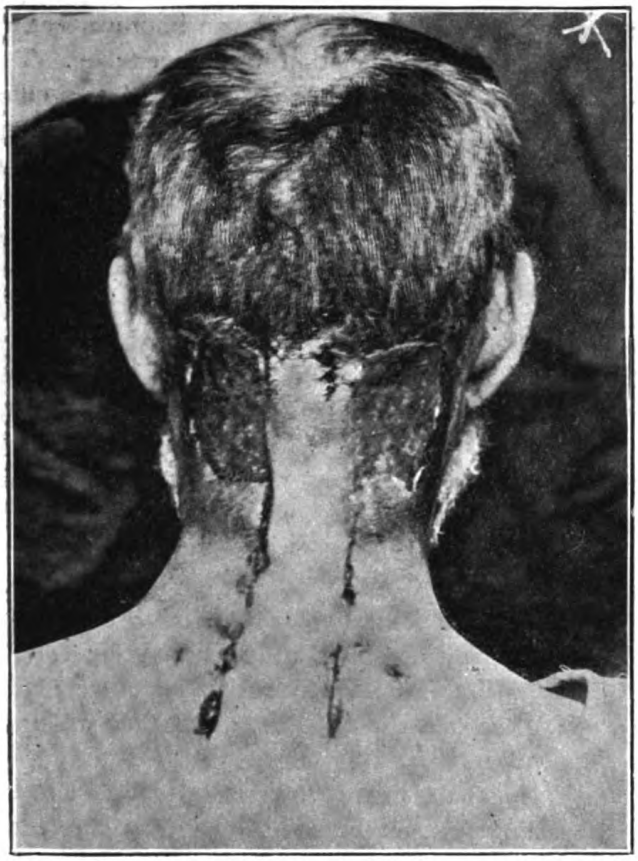


FIG. 11.—Ulcer, left by carbuncle, divided by long, stretched flap from back. Photographed after stitches were removed.

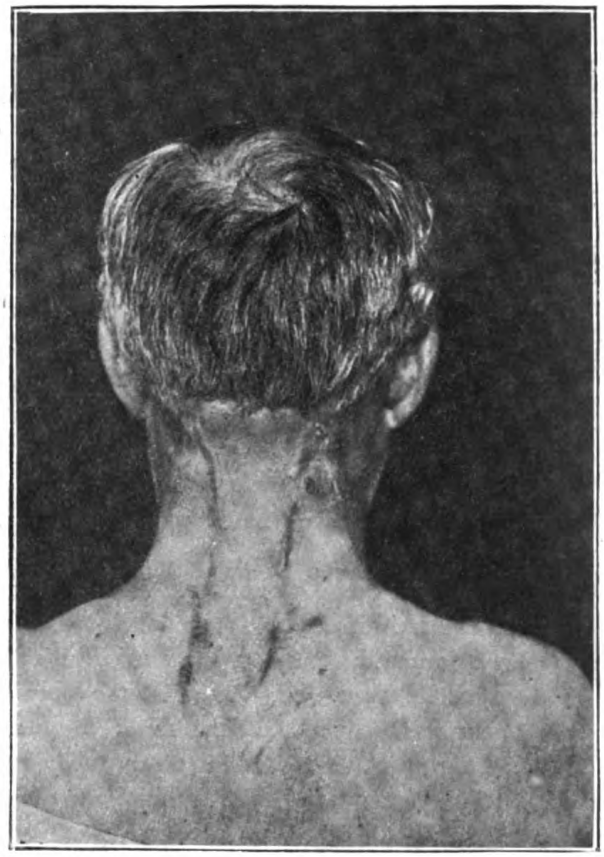


FIG. 12.—Same case as Fig. 11 after complete healing. Note that the upper part of the grafted flap is much stretched transversely.

FIG. 13.—To show thumb one month after transplanting graft from second toe containing nail with its matrix.

stroyed. The procedure is carried out as follows: The scar at the point of the finger is excised for the desired extent. A nail with its matrix is dissected out from the toe and inserted into the wound on the finger-tip. The graft should be shaped accurately and stitched all round. On the only two occasions this was tried, the plan was a success. Fig. 13 shows the result in one of the cases. The nail from the little toe is usually too small, while that from the great toe is too large. If the risk is considered justifiable the nail may be taken from the little finger, and its place again taken by the nail removed from the toe.

(3) *Bone grafting.*—(*a*) It is a well-known fact that bone grafts "take" better when accompanied by their periosteum, but this is not essential if a good piece of bone be chosen. Rib makes an excellent graft. Fig. 14 shows a case of hernia cerebri (traumatic and septic), and Fig. 15 the same after final grafting. Most of the herniated cerebrum was reduced and retained in position by packing together with an outer table bone-flap. The latter was only of temporary value, as it became absorbed to a large extent. The scar, after healing had taken place, was pulsatile and tender. The mental condition of the patient was good. It was considered that a proper support to the brain would be an improvement, so a bone-grafting operation was undertaken. The scar was excised, and the remains of the outer table graft removed along with it. Care was taken to leave the dura, or what represented it. Only a slight puncture was made through this with the escape of a drop of cerebro-spinal fluid. The bony margins of the gap in the skull were carefully exposed, and three pieces of ribs were jammed in across the gap. The remainder of the former bone plastic graft was utilised to fill in the lower end of the space. The skin was closed over the grafts without drainage. The result is shown in the photo. The scar was firm, and did not pulsate. From the patient's point of view there was a great improvement both in sensation and appearance.

(*b*) The treatment of a tubercular metacarpal bone usually results in the disappearance of the bone, with the subsequent shrivelling of the finger, which becomes a more or less useless and unsightly appendage.

Owing to the want of the metacarpal as a support the finger cannot be used. To reconstitute the lost metacarpal a section of rib was imbedded in the hand. A piece of the rib cartilage was placed against the phalanx, with the hope that it might

FIG. 14.—Case of hernia cerebri (traumatic and septic), taken under anæsthetic.

FIG. 15.—Same case as Fig. 14 after grafting of bone, etc.

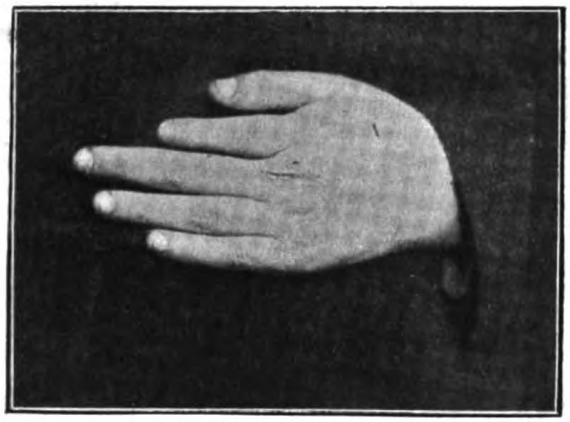


FIG. 16.—Case of tubercular dactylitis after grafting of rib to act as substitute for lost metacarpal bone, with result that finger's function was much improved.

assist in the formation of a new joint; but this section became absorbed, so the increase in length of the finger was ultimately not so much as appeared immediately after the operation, but the presence of the rib as a firm structure made the finger once more available for function (Fig. 16).

(4) *Fat grafting.*—The illustrations (Figs. 17 and 18) show the deformity left by a tubercular disease of the malar after healing and the result of grafting fat. The scar at the outer canthus was freed from the bone by a tenotome, and the skin raised up from the pit in the bone. To retain the skin in its new position a small piece of fat was excised from the thigh and packed into the cavity under the scar. A single suture closed the minute wound. The improvement was permanent. The second picture was taken two months after the graft.

(5) *Vein grafting.*—Sections of vein have been used in place of tendon, as a substitute for urethra, etc. The following is the plan of utilising a piece of vein as a covering to a newly sutured nerve. The saphenous is perhaps the best vein to select. Occasionally there is a little difficulty in locating the saphenous in the thigh. As a means of rendering its position demonstrable, the fingers of one hand should be lightly laid over the region of the vein while the other hand firmly and rapidly strokes up the leg, so as to send a sudden augmented flow of blood up the vein. The impulse in the vein is felt by the fingers placed over the thigh. About 2 in. of vein are removed, and threaded over a pair of sinus forceps (Fig. 19). By separating the forceps blades the vein is stretched to the desired extent. The forceps then grasps the end of the nerve, which is freed for the greater amount, and the vein tube is transferred over the nerve (Fig. 20). The nerve ends are then apposed and retained by two or three sutures. The vein section is then slipped over the junction and stitched to the nerve above and below the union (Fig. 21). By this means the sprouting nerve is contained in a tube; while fewer stitches are required, and the union will be all the more intimate and accurate. Adhesions are also avoided.

FIG. 17.—Depressed adherent scar after healing of malar tuberculosis.

FIG. 18.—Same case as Fig. 17, after elevation of scar, packing with fat taken from the thigh.

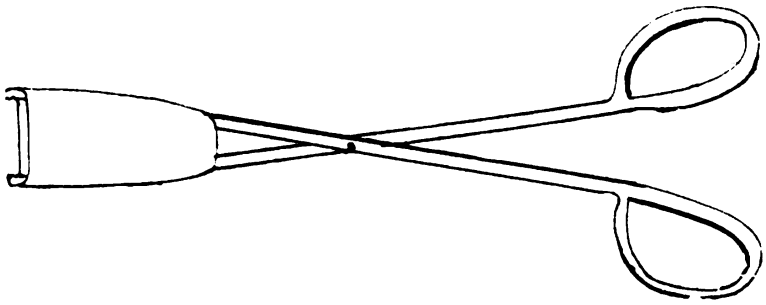


FIG. 19.—Vein stretched by sinus forceps.

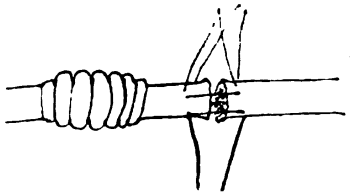


FIG. 20.—Three sutures placed but not tied to approximate nerve ends.

FIG. 21.—Vein section sutured to nerve after being spread out over junction.

REFERENCES.

(1) Craster, 'Journ. of Exper. Med.,' 1912, vol. xvi, p. 493.

(2) MacLennan, 'Glasgow Med. Journ,' August, 1908; Murphy, 'General Surgery,' 1909, p. 561.

P.S.—Since writing, the child depicted in Figs. 17 and 18 has been seen, now two years since fat was grafted; a difference between the right and left orbits was not observable.

November 17th, 1913.

The Treatment of Fractures near the Ankle-joint.—At a meeting of the Oldham Medical Society Mr. J. Griffiths, M.A., M.C., F.R.C.S., late Reader in Surgery in the University of Cambridge, gave an address entitled, "Fractures near the Ankle-joint." He treats these fractures in a very novel way. He maintains that any simple fracture near the ankle-joint, whetever its variety, can be perfectly treated by position alone without the use of splints. For this purpose it is necessary to lay the leg on its outer side upon pillows with the knee well flexed; the pillows are then secured to the leg with short lengths of bandage, and their weight keeps the limb in position. By this method the bones come into good apposition, the muscles are, and remain, relaxed, and thus cannot draw the fragments out of alignment. The patient is perfectly comfortable, and is encouraged to move his toes and ankle freely from the first; there is, therefore, no resultant stiffness or necessity for massage. Patients have been got up and induced to walk as early as the fourth day after the injury. Mr. Griffiths recommends that the period of rest shall not in any case exceed three weeks. When the time comes for getting up it is most important that the patient should be made to walk with the injured ankle well under the weight of his body, and not with the leg abducted as is the tendency. To secure this the sole of his boot is made about double the usual thickness on its inner side, bevelling to the unaltered outer side, with a slight thickening of inner side of heel. The result of the whole treatment is a perfectly straight and thoroughly useful limb. These principles are applied by Mr. Griffiths in the treatment of fractures in other parts of the body with satisfactory results.

LECTURES
ON
THE DIFFERENTIAL DIAGNOSIS OF CHRONIC GASTRIC DISORDERS.

Delivered at the London Hospital, August, 1913.

By ROBERT HUTCHISON, M.D., F.R.C.P.,
Physician to the Hospital, and Physician (with charge of Out-patients) to the Hospital for Sick Children, Great Ormond Street.

LECTURE II.

CONDITIONS WHICH SIMULATE GASTRIC DISEASE.

GENTLEMEN,—At our last meeting, you will remember, we were considering the general methods of investigating cases of abdominal disease; and I pointed out to you then that what you must always have present in your mind during your investigation is to try to elicit symptoms which point, on the one hand, to organic disease, or, on the other hand, to purely functional disorder. But before I consider, as I propose to do in two remaining lectures, the more exact differential diagnosis of the various organic and functional affections, I want to consider with you a group of conditions which may lead you astray: I want, indeed, to place before you a basket of "red herrings," any one of which may be drawn across the trail and lead you off the scent; conditions which are not directly or even primarily due to disorder of the stomach or intestine, but which may so closely simulate disease of these organs as to mislead both the patient and his doctor.

The simulation of gastro-intestinal disorders may be due, first of all, to *vomiting*, being a prominent symptom in the patient's complaint and in the history he gives you. There are many conditions not directly due to disorder of the stomach which present vomiting as an extremely important and troublesome feature of the patient's illness, and which he is apt, owing to vomiting being so prominent, to attribute to disorder of the stomach.

THE VOMITING OF PREGNANCY.

One of the first of those conditions which you will have to consider is the *vomiting of pregnancy*. I do not think any of you will have been long in practice before you will have made the mistake of regarding a case of vomiting of pregnancy as one of stomach disorder. It has happened to me on more than one occasion to make that mistake; it will certainly happen to you, as it happens to everybody who sees much of disease. I am not now talking of what is the commonest form of the vomiting of pregnancy, namely, morning vomiting, in which gastric symptoms are in abeyance for the remainder of the day. When the vomiting is limited in this way, you are of course more apt to think of the true cause. I am speaking now of those worst cases of vomiting of pregnancy, where the patient has sickness in association with every meal, where the sickness is attended by a certain amount of pain, as it often is, and where the frequent retching may give rise to some hæmatemesis. If you meet with a case where there is such a combination of symptoms, pain and vomiting, the vomiting relieving the pain, and the patient possibly bringing up a quantity of blood as the result of the strain induced by the retching, and if these symptoms occur, not in a young recently married woman, but in one who has already borne children, you are, and no doubt excusably, apt to forget the possibility of pregnancy as an explanation of the case. And yet all those symptoms may be due simply to the presence of gestation. It is usually sufficient to have the possibility of this error before your mind to enable you to avoid it, and if you look for the other signs of pregnancy you are not likely to make a mistake.

NERVOUS VOMITING.

The second form of vomiting which is very apt to simulate gastric vomiting is one which is closely allied in many respects to the vomiting of pregnancy, namely, "*nervous*" *vomiting*; some people style it hysterical vomiting. This is a condition which, I think, is met with in only the female sex, and in that respect it resembles the vomiting of pregnancy. In this condition the vomiting may be extremely persistent, and, indeed, that is one of the characteristic features of it, that it goes on and on for perhaps weeks, or even months; and the surprising thing about it is that, in spite of this apparent vomiting of everything she takes, the patient's health is wonderfully maintained, and she does not waste nearly as much as you would expect. A

further point is that the vomiting is practically independent of the kind of food the patient takes; she is just as much sick after taking liquids as after eating solids. In fact there is no rhyme or reason about the vomiting at all. Its most obvious feature is its extreme persistency. From these features alone in any given case you will be suspicious of a case of vomiting being nervous in origin, and, further, the history will often help you. There is frequently—I do not say always—a history of some emotional shock as the starting-point of the illness. One of the most marked cases I have had to deal with recently was that of a young woman who was much distressed by the death of her mother, to whom she was greatly attached, and her distress was still further increased by the fact that her father very shortly afterwards got engaged to a young lady who was a friend, and somewhat the junior, of the patient. It was this combination of circumstances which started in her the vomiting, and it persisted for several months. You will often find this form of vomiting also in young unmarried women who have nothing particular to do, and who live at home with their parents, particularly if the patient has a cranky and difficult mother. But remember you should carefully exclude every other possible cause before you arrive at the conclusion that the case is one of nervous vomiting. It is only by the exhaustive elimination of every other possibility that you are justified in finally coming to that conclusion. The mimicry of real gastric disease by this condition is so close that operations have been performed in some of these cases. In one case with which I had to do gastro-enterostomy had been performed, but, of course, without any benefit, for the vomiting in these cases is due to a central disturbance, and not to local disease in the abdomen at all.

Gastric Crises.

Closely allied in some ways to nervous vomiting is the vomiting of the *gastric crises* of tabes. Just as nervous vomiting occurs practically exclusively in women, so gastric crises are seen only in men. And in contra-distinction to nervous vomiting and that associated with pregnancy, which are persistent and continuous, the vomiting of gastric crises is characterised by its marked periodicity; it does not occur every day for weeks or months on end, but it takes place in bouts lasting two or three days, possibly longer, separated by intervals in which there are no gastric symptoms complained of. Further, the vomiting is marked by this—that, just as in nervous vomiting, the kind of food taken has no particular effect on it one way or the other. It is not usually attended by much pain, although there *may* be pain with it, but the most striking feature is that enormous quantities of highly acid fluid are brought up. You might be inclined to say that any person who is at all careful could not fail to recognise a case as one of gastric crises, but in that you would be wrong. Gastric crises are not so easily recognised as you might think, and for this reason, that they often develop before any of the ordinary signs of tabes make themselves manifest. Often—I would even go so far as to say usually—you will find that the knee-jerks are not absent in a case of gastric crises. You will often find, too, that the typical Argyll-Robertson pupil reaction is not present in its full degree, though there will probably be a sluggish reaction. Beyond that there may be nothing definite in the case to guide you in saying it is one of gastric crises, although, it is true, there may be a history of lightning pains, but often so vague as to be of very little assistance. If, however, you find marked periodic vomiting in a man, always be suspicious of gastric crises as the true explanation. If you investigate such a case further, you will often find that there is a history of syphilis, or the scar of a chancre can be made out; the Wassermann reaction will be positive, and there will be a lymphocytosis in the cerebro-spinal fluid. As I have already said, errors of diagnosis in regard to gastric crises are often made, and I have seen at least six cases where gastro-enterostomy had been performed on subjects of them, under the belief that the vomiting was due to ulcer. I have myself twice had occasion to have the abdomen explored because we were not certain that there was not organic disease of the stomach. In neither case was anything organic found, and so the abdomen was sewn up again. Both patients subsequently developed unmistakable tabes. So I want to impress upon you the fact that the simulation of gastric disease by gastric crises in men may be very close indeed, but here again the great safeguard is to have the possibility of the condition present to your mind.

Cerebral Vomiting.

The fourth form of vomiting in connection with the nervous system which may simulate gastric disease is *cerebral vomiting*, that which is due, for instance, to the presence of cerebral tumour. This form of vomiting is more apt to lead to error in the case of children, in whom cerebral tumour is not uncommon, and who are unable to give a good account of their symptoms otherwise—for instance, to tell you about headache, etc. A mistake, however, is really less excusable in the case of a child than in that of an adult, for the reason that gastric disorders which lead to habitual vomiting do not occur in childhood; children do not suffer from ulcer, carcinoma, etc., and so persistent vomiting in the child should not make you think of the stomach. Cerebral vomiting, it has to be remembered, has no relationship to the taking of food, but tends to come on spontaneously, though often the resumption of the erect posture after lying down is sufficient to determine an attack. Then, of course, you will look for other signs of cerebral disease, such as local paralysis, optic neuritis, and other indications of intracranial growth.

Next, we come to a group of cases of vomiting which are quite possibly due to catarrh of the stomach, but in which the gastric disorder is secondary to some serious disease elsewhere, although the occurrence of the vomiting is apt to make you think of the stomach alone, so that you may overlook the fact that it is a mere detail in the clinical picture.

Vomiting in Phthisis.

In this group I put first the vomiting of phthisis. Patients suffering from early pulmonary tuberculosis, in which the first complaint is often of gastric symptoms—nausea, vomiting, loss of appetite—will be brought to you under the belief that they have simple indigestion. I saw such a case only yesterday, the patient being a young man who was brought up by his father for supposed "indigestion." He had, as a matter of fact, early tuberculosis of one apex, and the vomiting was the kind which is associated with phthisis; it is quite likely due to catarrh of the stomach set up by tuberculosis.

Uræmic Vomiting.

Somewhat akin to these cases, although usually occurring in older people, is the vomiting which is associated with chronic nephritis, and which is, no doubt, uræmic in character. Here, again, as in the case of tuberculosis, the basis is probably a catarrh of the stomach. Still, the important point in the case is the kidney trouble, not the secondary gastric condition. These cases often occur in elderly persons, because it is especially in granular contracted or cirrhotic kidney that you meet with this kind of vomiting. And inasmuch as granular contracted kidney is also apt to lead to wasting and anæmia, these cases are very apt to be mistaken for gastric carcinoma. Here, again, if you will take the precaution which I pointed out to you in my first lecture, of examining the patient all over—all his organs—and not merely confining your attention to the abdomen, it is not likely you will fail to detect other indications of chronic kidney disease. Having made out such indications, you will be able to give the vomiting its proper place in the clinical picture; you will regard it as only an incident, and a comparatively trivial incident, in a case of grave disease elsewhere.

Uro-Kinetic Vomiting.

Last in this group of vomitings I put a kind which is closely allied to that of chronic nephritis, and which is sometimes called *uro-kinetic*, because it is due to disorder of the muscular apparatus of the bladder; it is a kind of vomiting, in fact, which occurs in patients who have some obstruction to the proper emptying of the bladder. You are, therefore, particularly apt to meet with it in elderly men who have enlarged prostates. Such a patient may come to you complaining, not of bladder symptoms at all, but of nausea, loss of appetite, sickness, and sometimes diarrhœa—a group of symptoms strongly suggestive of primary disease of the alimentary system, and yet which are really due to this failure of the bladder to evacuate its contents freely. I remember a very striking case of this sort which occurred here a few years ago in the person of a young man who had complained for some months of abdominal pain and vomiting. Sometimes the vomiting was so bad as to cause him to bring up some blood. He was treated as an out-patient for some weeks, under the belief that he had a gastric ulcer. His symptoms persisted, and eventually became so bad that he was admitted into one of the wards,

and there it was found, on going round one day, that his bladder was distended up to the umbilicus. The patient then said—what he might have told us before—that he had a difficulty in emptying his bladder, owing to the presence of a stricture; and it was when the greatest difficulty in emptying the bladder occurred that his stomach symptoms were worst. The stricture was dilated, so that the bladder could be freely emptied, and from that moment the patient lost his gastric symptoms. In arriving at your diagnosis in such a case the presence of dilatation of the bladder will often be of help. You will often find it as a long and narrow tumour in the hypogastrium. When you find that, and there are other signs of prostatic enlargement, and the patient complains of dyspeptic symptoms, always be strongly suspicious that the dyspeptic symptoms are secondary to the condition of the genito-urinary apparatus.

Migraine.

There are two other conditions accompanied by vomiting which may simulate abdominal disease which have still to be mentioned. One is *migraine*, a condition which the patient is very apt to describe as "dyspepsia." I do not, of course, refer to the classical examples of migraine, which begin with violent headache—hemicrania—and after a few hours end up with sickness. I do not think anyone would be likely to mistake such a case for one of primary gastric disorder. I am referring now to atypical cases in which headache is a very minor symptom, and the vomiting a very prominent one; where the patient has vomiting almost from the outset, and where it may go on for some hours, even days, and in which, as in all severe and prolonged vomiting, the patient may bring up some blood. These are cases in which gastric disease is apt to be simulated. Here, again, the marked periodicity of the attacks makes the case unlike one of true gastric disease. The attack comes on, lasts a varying number of hours, and then ceases, the patient remaining free from gastric troubles until two or three or more weeks later, when he gets another attack; and in that periodicity they more resemble gastric crises. This form of migraine, so far from being, like gastric crises, confined to the male, is common in women, though it may be met with in both sexes; and you make your diagnosis partly from the periodicity of the attacks, and partly from careful inquiry respecting the head symptoms which accompany them. When the patient is cross-examined, he will usually admit the presence of headache, of the typical hemicrania variety, or of some of the ocular symptoms which occur in an attack of migraine, although the vomiting has become so prominent a feature that he has almost forgotten the existence of the headache.

Cyclical Vomiting.

Finally, there is a form of vomiting which is met with only in children, the so-called *cyclical vomiting*. This condition also is apt to be regarded as a dyspepsia, although really it is not a primary gastric trouble. I admit that the pathology of this form of vomiting is obscure, and it is not quite certain what part the liver and the nervous system, respectively, play in producing it. Many of the cases are extraordinarily like those cases of aberrant migraine which I have just described, and it is interesting that when children who suffer from cyclical vomiting get past the age of puberty, it is apt to disappear, and its place to be taken by typical migraine. There is, therefore, no doubt a close relationship between cyclical vomiting and migraine. In cyclical vomiting there is no headache, or if it occurs, it is secondary to retching and straining. The characteristic feature is that at intervals, often periodically, the patient is attacked by violent uncontrollable vomiting, which lasts some hours, or a few days, and is accompanied by obstinate constipation. In the urine, in these cases, you will find aceto-acetic acid and acetone, one feature of the attacks being the presence of a marked acidosis, but whether that is the cause or the consequence of the attacks is a disputed point. When you find these features in a child below the age of puberty, particularly a girl, it should make you think of cyclical vomiting, and not of a primary gastric condition. Here again the cases may be very deceptive in their mimicry of abdominal disease. I have known them, owing to the marked, obstinate, and almost insuperable constipation which accompanies them, to be mistaken for intestinal obstruction. More than once the abdomen of such a child has been opened, under the belief that the patient had acute intestinal obstruction—a mistake which it is very dangerous to make, because the opening of the abdomen implies the giving of an anæsthetic, and there is

no worse subject for an anæsthetic than these patients, and there is no worse time for giving an anæsthetic than when an attack is on, for the condition known as delayed chloroform poisoning is peculiarly apt to develop in them, and is not uncommonly fatal.

Pain in the Abdominal Wall.

And now I want to pass to a group of cases in which it is *pain* referred to the abdomen which makes the patient or his doctor believe him to be the subject of disease of the stomach or intestine. And first the pain which is so misinterpreted may be situated in the abdominal wall, using that term in its widest sense. The pain of spinal caries has been mistaken for dyspepsia, particularly in children. Here, again, the mimicry is extraordinarily close. I was last month concerned with a very able general practitioner and with two surgeons in seeing a young man who complained persistently of pain in the back. He had pain in the lower dorsal region, with definite rigidity on moving and bending. He also had a slight rise of temperature. There was a marked family history of tubercle. After a very careful examination, on more than one occasion, we decided it was probably a case of spinal caries—a decision which was extremely important, for the patient was an officer in the army, and if we had definitely decided it was spinal caries it would have meant his resigning his Commission. He was radiographed and the result of that was negative. To cut a long story short, I may tell you that after much deliberation his abdomen was opened, and it was found that he had a subacutely inflamed appendix, and removal of that brought his symptoms to an end. Here, then, was a case in which it was not spinal caries simulating abdominal disease, but the position was the other way round, showing—and this is the point—that conditions in the abdomen may cause pain in the abdominal wall and spine owing to the intimate association which exists between the abdominal organs and the nerve-roots which emerge from the spinal cord.

Then you may find such conditions as lateral curvature, which, by pressure on nerves, may cause pain referred to the abdomen, which, for a long time, may lead to the belief that the patient is the subject of abdominal disease.

Then there is fibrositis of the abdominal muscles, so-called "muscular rheumatism," which causes pain in the muscle, with tenderness on palpation. There may be such fibrositis in the upper parts of the bellies of the recti, or it may occur in the right iliac fossa, simulating appendicitis. Such a condition is not so very uncommon; it is probably commoner than it is generally believed to be, and the characteristic diagnostic feature of it is that the pain is very much affected by movement. When the patient strains the affected muscles, such as getting up in bed without help, he gets pain. If you pinch up the muscles laterally, so as not to produce deep pressure, they are tender, showing that the pain does not originate from the underlying structures, whereas true abdominal pain, due to disease of the viscera, is increased by pressing back on those viscera.

Epigastric Herniæ.

Among the affections of the abdominal wall which may deceive you, one should mention epigastric hernia. Every now and then you will meet with patients who have a small protrusion forming a little lump in the epigastrium, which is really a protrusion of the fat of the greater or lesser omentum through a gap in the linea alba. Such a hernia is apt to cause pain, and sometimes vomiting, by dragging on the stomach or its attachments, and so may lead to an erroneous diagnosis of primary disease of the stomach. Here, again, if you are on the lookout for the condition you will hardly fail to recognise it.

Gall-stones.

Passing now to conditions inside the abdomen which may simulate disease of the stomach, one would speak, first of all, of pain in the gall-bladder. Gall-stones, when confined to the gall-bladder, do not, of course, show themselves by attacks of biliary colic; it is only when they get down into the duct that they cause that. Nor, when they are confined to the gall-bladder, do they cause jaundice. Hence, the two classical symptoms of gall-stones, namely, biliary colic and jaundice, are conspicuous by their absence when the stones remain in the gall-bladder. As a consequence of this it is apt to be forgotten that pain in the abdomen, in spite of the absence of typical jaundice or colic, may be due to gall-stones, and pain of this origin is often attributed to disease of the stomach, particularly, perhaps, to ulcer. The difficulty of diagnosis is

rendered still greater by the fact, to which I have already referred, that these gall-stone pains have often a definite relation to the taking of food. They have a relation, first of all, to the kind of food taken, the attacks being definitely brought on by certain articles, and not by others; and secondly, the pain often has a definite time-relation to meals; it will come on an hour or an hour and a half after taking food, or sometimes it is a hunger-pain—that is, coming on late after a meal, and relieved by eating. When you have a patient complaining of such pain with a definite relation to meals and the kind of food eaten, and with no history of jaundice or biliary colic, who can wonder if disease of the stomach is diagnosed? There are, however, one or two points about the pain which should make one suspicious. In the first place, it often wakes the patient up in the middle of the night. Secondly, it is attended by a feeling of flatulence and distension; and, thirdly, vomiting is usually absent, which would hardly be the case in gastric ulcer. But when all is said and done, the diagnosis of stomach symptoms due to gall-stones is not always an easy matter, and sometimes it is only by prolonged watching of the case that you can come to a right conclusion. Sometimes the most you can say is that you are not sure about the presence of gall-stones, but that you are sure the patient has some organic disease, and therefore the case is one which calls for an exploratory operation. Many times that is as near as one can get to the diagnosis.

Renal Calculus.

Then there is a group of conditions associated with the kidney which are apt to be mis-diagnosed. And first of all stone in that organ. Here, again, if you are dealing with a typical case where the pain begins below the last rib, and passes round the costal margin and down into the groin, and is attended by definite urinary symptoms, there is no likelihood of your making a mistake. But the symptoms of the stone in the kidney do not always pursue that classical form. Medicine would be an easy matter if all diseases "played the game," and, I may say incidentally, it would be a much less interesting study. It is because stones do not always produce the regular orthodox symptoms that error is apt to arise in diagnosis. A stone may cause vague pains in the right or left hypochondrium, which the patient sometimes fancies has a definite relationship to meals. Not long ago, for example, I saw a gentleman who had definite hunger-pain, and it came on in such bouts that it seemed certain he had duodenal ulcer. Exploration was carried out, but the surgeon did not find a duodenal ulcer. He, however, found an unhealthy appendix—as they always do—and removed it. But the patient's symptoms recurred, and one day, after some violent jolting, the patient had a typical attack of renal colic. An X-ray examination revealed a stone and it was removed. After the operation he lost his hunger-pain.

Movable Kidney.

A movable kidney is a common source of error. I am not referring at the moment to cases of "Dietl's crisis," as it is called, but to the cases of movable kidney in which the kidney, by dragging on the stomach at its pyloric end, can produce symptoms which are strongly suggestive of primary gastric disease. And of course—unfortunately for the diagnosis—cases of movable kidney often occur in association with genuine gastro-intestinal disease, gastroptosis, for instance, which is almost always accompanied by movable kidney. Gall-stone cases are often apt to be accompanied by movable kidney, and, therefore, when you find there is a movable kidney you must not necessarily conclude that it is the cause of the patient's symptoms. You must, however, have your mind open to that possibility, and see whether by fixing the kidney, by means of a suitable truss, or by a belt, you cannot make the symptoms disappear.

Aberrant Renal Vessel.

Still more difficult are the cases which are due to stricture of the ureter by an aberrant renal vessel. An aberrant renal vessel, by constricting the ureter, causes periodic recurring abdominal pain, which is sometimes so severe as to lead to vomiting. And such cases are extremely apt to be misunderstood, because you say to yourself, not unnaturally, that constriction of the ureter is bound to be manifested by urinary symptoms. But the surprising thing is that these patients do not have urinary symptoms; their sole symptom is pain, and sometimes a feeling of throbbing in one or other side of the abdomen. These cases are apt to be mistaken for all sorts of other conditions. I know a doctor, a former

student at this hospital, who was the subject of severe recurring abdominal pain. Being a doctor he was on the lookout for evidence to show which organ was involved, but was quite unable to arrive at any conclusion. A surgeon was called in, and he removed the appendix. The symptoms, however, recurred, and it was supposed to be a case of gall-stones. The gall-bladder was explored, but there were no stones. Then it was found, I think at the second operation, that he had an abnormal renal vessel which was constricting the right ureter, and causing bulging above it. That was put right, and the attacks disappeared. Here, then, was a man who had been trained to observe cases of abdominal pain, and yet, in his own person, was unable to say whether it was the kidney which was the seat of his trouble. It serves to show how deceptive these cases are. The patient in these cases of compression of the ureter is almost always a male. The attacks begin early in adult life, in adolescence usually. They are distinctly periodic and they consist in severe pain, often with throbbing, usually somewhere about the loin or the lateral aspect of the abdomen, but usually with an absence of urinary symptoms. Catheterisation of the ureters and an X-ray examination after the injection of collargol will enable one to obtain objective evidence of the abnormality, but all physical examination short of such means yields a negative result.

Appendix Dyspepsia.

Passing on to consider other organs, the presence of disease in the appendix may deceive you into the belief that the patient is the subject of gastric disease, particularly of gastric ulcer. In the short time at my disposal it is not possible to go fully into the question of "appendix dyspepsia," but there can be no doubt at all that chronic disease of the appendix does sometimes simulate most closely gastric disease, by producing pain in the epigastrium, vomiting, and even hæmatemesis. But at the same time it it also possible that the appendix is blamed now more often than it should be for things which go wrong inside the abdomen. It is a kind of abdominal scapegoat: anything unpleasant which is felt in the abdomen and which can be attributed to nothing else is thought to be due to disease of the appendix. Now, it is a matter of extreme difficulty to say whether there is chronic disease in the appendix or not. Personally, I do not like to accuse the appendix unless there is definite tenderness in the right iliac fossa, but there are many people who recommend operation on evidence far short of that. All I would say now is, that you must always have your mind open to the possibility of the appendix being at at fault. Perhaps, however, this admonition is hardly necessary at the present time, because most people's minds seem to be too much open about it. There are one or two points about the pain in appendix dyspepsia which may help to keep you right. It is usually more constant than the pain of gastric ulcer, it is on the whole less affected by food and more by exertion, and it is usually at some time felt in the lower abdomen, even although it may spread to the epigastrium.

Last among the cases of pain which may simulate gastric disorder there is a group which is due to disease of the vascular system.

Angina Pectoris and Abdominalis.

First among these is angina pectoris. Here, again, I do not mean the classical full-blown angina pectoris which is described in the books; I mean the minor forms of angina. These may simulate cases of gastric disease for this reason: that they exhibit two essentially stomach symptoms—pain, referred to the lower end of the sternum or the epigastrium, and the eructation of wind. But the characteristic thing is that the pain is related not to food, but to exertion. If a patient tells you that the pain comes on when he exerts himself, particularly if he does so soon after taking a meal, then you should look out for the other evidence of angina pectoris. Still more deceptive are the cases of angina abdominalis, the true nature of which is not quite clear, although it is probably due to spasm of some of the abdominal arteries. The chief symptom is a crampy pain in the abdomen, possibly attended by other symptoms, such as diarrhœa. In these cases, again, the pain is related more to exertion than to anything else. The patient is usually elderly; often he is one who has smoked a good deal; he has a high-tension pulse and degenerate arteries, and, on the administration of vaso-dilators, or of the drug diuretin, the symptoms disappear.

Abdominal Aneurysm.

Lastly, among vascular troubles, mention should be made of abdominal aneurysm, which sometimes leads to error, because it produces pain in the upper abdomen. Abdominal aneurysm is, of course, rare. Curiously enough, the subjects of it are usually old soldiers, and particularly men who have seen service in India. It is extraordinary that nearly all the cases of abdominal aneurysm I have seen here have been in men who have been in the Indian Army. The pain of an abdominal aneurysm is almost always worst on lying down. That is very unlike the behaviour of most forms of abdominal pain, for the majority of them are relieved when the patient lies down. The pain of abdominal aneurysm is also felt as a boring pain in the back, and on careful examination you will find an expansile pulsating swelling deep in the epigastrium. On examining by means of X rays you will make out the existence of aneurysm.

Eructatio-Nervosa.

I have spoken of cases where you are deceived by the presence of vomiting or of pain; and it is now necessary to mention a condition where you are deceived by the occurrence of flatulence. The condition I am about to speak of is one in which the patient is always wrong in his own diagnosis; he thinks he has got dyspepsia, but in nine cases out of ten he has not. The condition is called eructatio-nervosa, or aërophagia, or air-swallowing. And the characteristic of it is simply excessive flatulence. The patient brings up enormous quantities of wind, and, in consequence, is supposed to be suffering from flatulent indigestion. He is dieted for flatulence, and given stomach mixtures without success, and the real fact that he has an affection of the nervous system is overlooked. Eructatio-nervosa is not an affection which, as is commonly taught, occurs only, or even mainly, in women. It is true that the most marked examples do occur in women about the menopause, but the majority of cases of it I have seen have been in men, many of them young men who were otherwise healthy. The flatulence occurs independently of food and even when the stomach is empty, so that the patient, on getting up in the morning, may bring up enormous quantities of wind. The characteristic point about this affection is that the patient can eructate to order; if you tell him to bring up wind, he does it; he has a difficulty, sometimes, in stopping himself. When you can get a patient to eructate to order, you can be almost certain that the case is not one of dyspepsia, but a neurosis; the nervous mechanism controlling the œsophagus and the cardiac sphincter being disordered, so that the patient can suck down large quantities of atmospheric air, and bring them up again; sometimes the process will go on until he feels almost exhausted. It is usually sufficient in these cases to explain to the patient what he is doing, and immediately he gets cured. The existence of the habit depends on its being subconscious, and it is like so many subconscious acts—when you begin to think about it, it stops.

The next lecture will be devoted to the differential diagnosis of organic affections of the stomach.

November 17th, 1913.

Teeth present at Birth.—P. Balard and C. Commes state that during a period of 10 years among 17,578 children born in the Maternité de Paris, only 3 had teeth at birth, and during the last twenty years at the Bordeaux Clinique d'Accouchements there were only 2 cases among 10,000 births. In 2 cases a lower median incisor was seen on the day after birth, and the following day its fellow appeared. As suckling was thereby rendered painful to the mother the teeth were extracted. Neither child presented any other anomaly and their family history was negative. In a third case the two lower median incisors were present at birth, but fell out spontaneously three weeks later. The child also had a congenital cyst of the iris. Microscopical examination of the teeth of the first two cases showed they were of the nature of a teratoma.—*Brit. Journ. Child. Dis.*, x, p. 464.

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THE USE OF CELLULOID SPLINTS IN THE TREATMENT OF ACUTE CASES OF POLIOMYELITIS AND OTHER NERVOUS DISORDERS.*

By FREDERICK E. BATTEN, M.D., F.R.C.P.,

Physician to the Hospital for Sick Children, Great Ormond Street, and to the National Hospital for the Paralysed and Epileptic.

THE object of my lecture to-day is to put before you a simple, effective and cheap method of splinting cases of acute poliomyelitis so as to prevent deformity and hasten recovery. The principle "that a paralysed muscle should be placed in the position of rest" is, I think, generally accepted.

There are, of course, numerous methods by which such rest can be secured. Some medical men think that simple rest in bed is sufficient, others apply a splint so as to keep the limb in the normal position, and others, again, place the limb in a fixed position so that there is complete relaxation of the paralysed muscle and hyperextension of the antagonistic muscles. The actual line of treatment must vary in different cases, and it is probable that each of the above methods is suitable in individual cases. The method which I advocate to-day is the application of a celluloid splint, and I do so because I think it is effective, can be made by, or under the direction of, the medical man under whose care the patient is, and is not expensive.

The celluloid splint may be said to have a triple object: first, of keeping the muscle at rest in a position of relaxation; secondly, of preventing deformity; and thirdly, of enabling the patient to walk, and thus applying a physiological stimulus to aid the recovery of affected muscles.

* Lecture at the Polyclinic.

These splints were first used by M. Calot,* of Berck-sur-Mer, and introduced into this country by Mr. Gauvain† in the treatment of tuberculous disease of the bones. We are indebted to Mr. Gauvain for many of the technical details in the making of the splints. I have used them frequently during the past two years‡ in the treatment of cases of acute poliomyelitis, and they have proved of the greatest service in preventing deformity and hastening recovery.

The splints are light, can be worn both during day and night, are easily removed for washing and massage, or for any other lines of treatment that may be considered necessary, and are easily reapplied. They can be made sufficiently strong to support the weight of an adult, and are not readily broken. They weigh comparatively little—those for a child being about 10 to 14 oz., those for an adult from 24 to 32 oz.

There are three processes involved in making these splints:

(1) The taking of the "negative" cast of the patient's limb.

(2) The making of the "positive" cast from the negative.

(3) The moulding of the splint on to the "positive."

These three processes will be described in detail, but it is the first which it is essential for the medical man to perform, for unless the limb is in the right position the splint will be useless. The two latter processes can be performed by an instrument-maker, but it is quite easy for the medical man himself or anyone gifted with a little manual dexterity to make them. Some of my most successful splints have been made by the "Sister" in charge of my ward.

(1) The Taking of the "Negative" Cast.

For this purpose a strip of lead ¾ in. wide, and 2 to 3 ft. in length, some oil, a strong knife with a sharp point, and some plaster-of-Paris bandages are required.

It is important to have the right kind of plaster-of-Paris bandages; the ordinary surgical bandages supplied by instrument-makers are quite unsuitable. The bandages should be made of book-muslin, should be 4½ in. wide, and 4 to 6 yds. in length, should be loosely wound, and not too heavily loaded with plaster.

The limb of which the cast is required—let us say for the sake of example the leg—is oiled, having previously in the case of a hairy individual been shaved. The strip of lead is oiled and adjusted to the front of the leg.

The leg is now held in the position required; there should always be slight flexion of the knee and the foot should be held carefully at right angles to the tibia. It is important not to hyper-extend the knee or to dorsiflex the foot, for it will be found that the child cannot walk with the leg in such a position. The plaster-of-Paris bandages are placed in warm water, and after being squeezed out are wound round the limb, starting from the foot and working up the thigh. It is best to take each bandage continuously up from the foot to the thigh; if this is done, there is but little liability for the cast to crack transversely when taken off the limb. For a child six to eight years of age, about four to five bandages are usually required. When the plaster has set sufficiently, which usually takes place about three to five minutes after the completion of the application, a cut is made down the anterior surface on to the lead. This incision must be complete and all fine threads must be divided, for unless this is done the cast is liable to be spoilt by portions of the bandage being dragged out of place. The cut edges of the plaster are now separated and the lead removed through the incision. The cut edges are now further separated sufficiently to allow the leg to be removed. This is best effected by catching hold of the toes and dragging them forward, whilst an assistant separates the edges of the cast and pushes it backwards. When the cast has been removed the cut edges are carefully approximated and a single layer of plaster-of-Paris bandage is wound round the cast, the toe-end being completely covered in. The negative cast is now complete and is allowed to dry.

(2) The Making of the Positive Cast.

In twelve to twenty-four hours the "negative" cast is dry, and from this the positive is made. The negative cast is oiled on the inner side, a bamboo cane is placed down the centre, and after plaster-of-Paris has been mixed in the usual

* Calot, 'Orthopédie Indispensable,' Paris, 1913.

† Gauvain, 'Brit. Med. Journ.,' 1913, i, p. 1200.

‡ Batten, 'Lancet,' 1912, ii, p. 80.

manner, it is poured into the cast. This is allowed to set, and after twelve hours an incision is made down the anterior surface, the negative peeled off and the positive removed.

(3) The Moulding of the Splint on the "Positive."

For making the splint the following articles are required: stockinnette, book muslin, solution of celluloid in acetone (11 oz. of celluloid to 160 oz. of acetone, to which should be added a solution of 3 oz. of calcium chloride dissolved in 2 oz. of hot water; this should be added while still hot to the celluloid solution and well shaken up. The product is "Gauvain's solution"), celluloid varnish (cellulose triacetate 1 part, acetone 5 parts), some leather for binding the splint and some boot hooks.

Method.—A layer of stockinnette is first fixed over the cast and sewn on. Over this is placed a layer of book muslin cut so as to conform roughly to the shape of the limb. On this is painted a coat of the celluloid solution; another layer of book muslin is applied, followed by another coat of celluloid and so on, until fourteen to eighteen coats have been applied. It is well to allow one coat to dry before applying another. About the ankles and knee it is well to strengthen the splint by three to four extra layers of muslin and celluloid. After the final coat has been laid on a coat of celluloid varnish is applied. When this has been done two incisions are made down the front of the splint and a strip about ½ in. in breadth is removed. This allows the positive to be taken from the interior of the splint.

The splint is now fitted on the patient. If the cast has been well made it usually fits perfectly; some small amount of the splint may need to be cut away about the knee and ankle, so as to allow of easy application and removal.

The splint, after being bound round with leather and hooks inserted, is ready for wear.

In early and acute cases, when the patient is still in bed, the splint is applied and worn both day and night, being removed only for massage and passive movements twice a day. The splint is worn next to the skin, which should be dusted with boracic or other powder. It is inadvisable for the patient to wear a thick stocking inside the splint; the thickness of the stocking displaces the limb and the splint will no longer fit well. If the splint seems to cause pressure at any point it should be stretched and manipulated so as to relieve this pressure. It is inadvisable to put wool or leather inside the splint to avoid pressure; if the pressure on the malleolus cannot be relieved by manipulation of the splint it is best to cut a hole in this region and to pare off the edges.

At the end of the first month, after the onset of the disease, it is usually possible to get the patient up, and the splint enables such patients to walk without any liability to the production of deformity. Over the splint the child wears an india-rubber sand-shoe, and for most purposes this is quite satisfactory, but a boot can be worn over the splint if desired. The earlier attempts at walking must be very carefully performed, but with the aid of a walking apparatus the patient rapidly gains strength, and in many cases is soon able to walk alone (Figs. 1, 2, 3).

Class of Case Suitable for Splinting.

Almost any paralysis of the lower limbs is suitable for support with celluloid splints. The cases which may be considered unsuitable, and in which no good result is to be obtained, are those with complete loss of power in the psoas and iliacus group and in the glutei. Those cases in which the back, lumbar and the abdominal muscles are much affected are also unfavourable; some cases, however, where there has been considerable affection of the dorsal and abdominal muscles and but little affection of the lower limbs are greatly helped by a celluloid spinal jacket. The extent of the splint must vary according to the degree of the paralysis. If the paralysis in the leg is at all extensive, it is best to make the splint encompass the whole leg as high as the tuber ischii behind, the trochanter on the outer side, and the ramus of the pubes on the inner side. If the paralysis is limited to the muscles below the knee a splint as far as the knee is all that is required.

The line of treatment adopted in acute cases of poliomyelitis is complete rest in bed for at least four weeks after the acute onset, the administration of urotropin, the washing out of the mouth and the nares with a solution of ·2 per cent. permanganate of potash. If pain is severe it is generally relieved by aspirin, but in some cases small doses of morphia are essential. Some form of temporary splint should be applied within the first fourteen

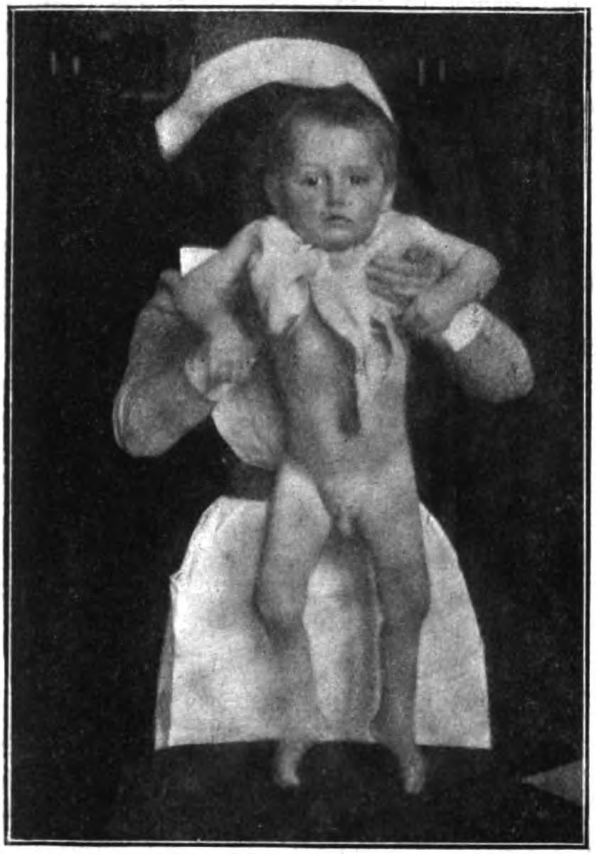

FIG. 1.—Illustrates an almost complete flaccid paralysis of both lower limbs in a boy, æt. 3 years.

FIG. 3.—Illustrates the form of walking apparatus used in encouraging children to make an attempt to walk.

FIG. 2.—The same boy in celluloid splints reaching from the foot to the groin. Note the flexion of the splint at the knee and the shoes worn over the splint.

days if there is any tendency to deformity. As soon as pain has subsided a cast of the limb should be made, and the celluloid splint moulded thereon; this should be applied and worn at first both day and night, being only removed for washing the limb and for treatment.

In those cases in which there is paralysis without pain, massage may be begun fourteen days after the acute onset, but in many cases it is not possible to begin before the end of the fourth week. After the end of the fourth week the child should be allowed first to sit up, then to assume the erect position and gradually encouraged to make an attempt to walk. A walking apparatus on wheels is of great assistance in starting and aiding these children to walk (Fig. 3).

It is essential to keep the limbs warm. Putting the muscles into physiological action greatly helps t [illegible]

Celluloid splints can be worn for six to twelve months whilst recovery is taking place, and as return of power occurs can be gradually discarded. In the later stages of the disease, if much paralysis persists, steel instruments are probably preferable to celluloid splints.

This form of splint is also useful in dealing with cases of paralysis of the arm; the biceps can be kept at rest in a flexed position, and, if necessary, the deltoid, by abducting the arm from the body. These cases of paralysis of the muscles supplied by the fifth cervical root are, however, most difficult to deal with, and the results obtained by any method are far from satisfactory (Figs. 4 and 5).

The Use of Celluloid Splints in Nervous Disorders other than those produced by Poliomyelitis.

Although I have used celluloid splints to a greater extent in the treatment of paralysis follow-

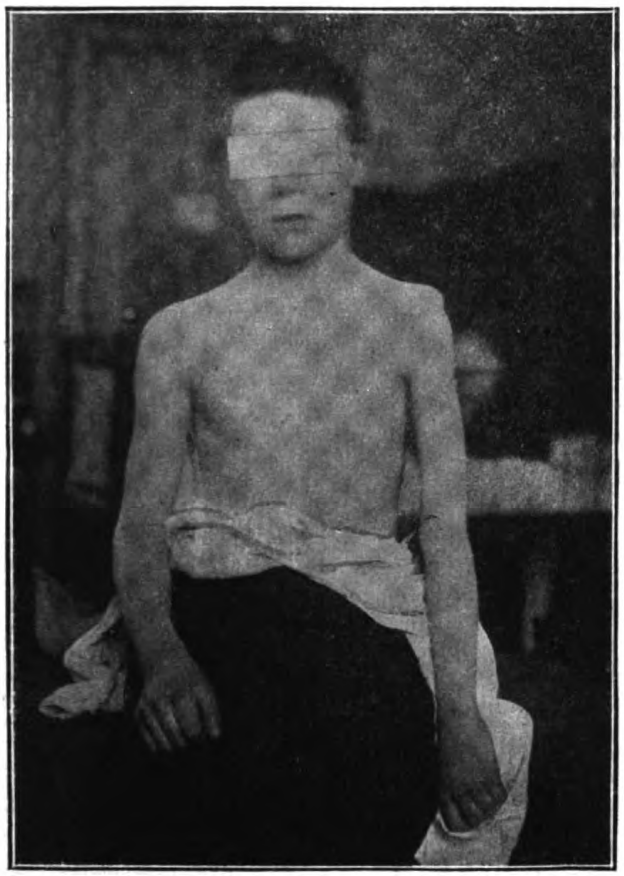

Fig. 4.—Shows a boy with complete paralysis of the deltoid and biceps on the left side.

Fig. 5.—Shows the same boy with the arm placed in a flexed position in a celluloid splint. A nerve anastomosis in this boy failed to give any good result.

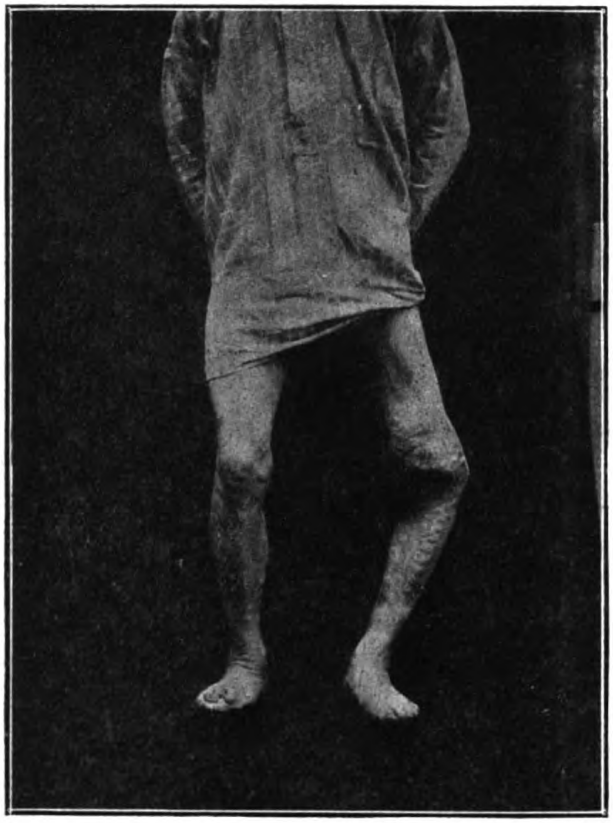

FIG. 6.—Shows the appearance of a knee of a tabetic patient affected with Charcot's disease.

FIG. 7.—Shows the same knee splinted with a celluloid splint.

ing poliomyelitis than in any other condition, yet the splint has been of service to me in cases of *hemiplegia*, with contraction and inversion of the foot, in *contracted conditions of the hand* and *in wrist-drop*. Again, in cases of *peripheral neuritis* with foot-drop a light celluloid splint is of service in keeping the foot in the dorsi-flexed position whilst recovery is taking place. In cases of tabes, with *Charcot's disease of the knee-joint*, the celluloid splint is of great value, for it not only enables the patient to walk without the risk of the knee suddenly giving way, but the fixation of, and even pressure around the joint tends to prevent the extension of the disease and the effusion which often takes place into and around the joint.

It is important, before taking the cast of a knee-joint affected with Charcot's disease, to allow the patient to rest for some few days in bed, so that the œdema and swelling of the joint may subside. Unless this is done, the splint is liable to be too loose after having been worn for a short time (Figs. 6 and 7).

CONCLUSION.

It may be said that in the treatment of acute cases of poliomyelitis a celluloid splint is of the greatest service, for it is light, can be worn day and night, keeps the limb in good position, and can be used either while the patient is at rest in bed or walking about.

The splint is also of great service in other affections of the nervous system, and especially in affording support to a joint affected with Charcot's disease.

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LECTURES
ON
THE DIFFERENTIAL DIAGNOSIS OF CHRONIC GASTRIC DISORDERS.*

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LECTURE III.

ORGANIC DISEASES.

IN my last lecture, gentlemen, I was considering with you some conditions which are apt to simulate gastric disease, and which may therefore lead you at the outset of your examination of the case into error. And I want now, having cleared away those conditions, as it were, to deal in the remaining lectures, first, with organic affections and their differential diagnosis; and secondly, in the last lecture, with functional affections.

To-day, then, I shall speak of the organic affections of the stomach. These are not at all numerous. For practical purposes they comprise gastritis, or, if you prefer the term, gastric catarrh; ulcer, including therein both gastric and duodenal ulcer; carcinoma; and organic dilatation. There are, in addition, two conditions which are, as it were, on the borderland of organic disease, so that there is some doubt as to whether they should be regarded as organic or as functional. I refer to gastroptosis and a condition which we shall call just now for convenience hæmorrhagic gastralgia. I shall not deal with these to-day. They will best be considered, I think, when we come to the functional disorders, because gastroptosis is almost always associated with more or less deficiency of gastric tone, which is an important functional disorder of the stomach, and hæmorrhagic gastralgia we may consider along with the question of gastralgia in general—that is to say, as a disorder of the sensory functions of the stomach.

GASTRITIS.

You will remember, as I have repeatedly pointed out in the last two lectures, that organic diseases of the stomach all tend to be characterised by certain symptoms which mark them off from the functional affections. These symptoms are more or less severe pain, vomiting and wasting. All of these symptoms need not be present in any one case, but they are the sort of symptoms which indicate organic disease. Curiously enough, gastritis or gastric catarrh is the one exception to that statement; it is an organic affection of the stomach which is not characterised by pain, nor, as a rule, by vomiting, although nausea is usually present, nor yet by wasting, at least not in any marked degree. And it is also the one organic affection of the stomach which is not suitable for surgical treatment. I have told you that the organic affections of the stomach, as opposed to the functional, are, or in my opinion at least ought to be, surgical diseases; but to this gastritis is an exception. Catarrhal conditions of the stomach are therefore much more akin, both in symptoms and treatment, to functional than to organic disease, and it is with functional conditions that they are most frequently confounded. A very common mistake is to diagnose cases of functional dyspepsia as "gastric catarrh"; but I want to impress upon you the fact that in reality gastritis is a comparatively rare affection. Functional disorders of the stomach are extremely common; true gastritis is not common; and I may add that it is one of those conditions which cannot be diagnosed with certainty except by the use of the stomach-tube. Fortunately there are not many stomach affections of which that can be said; but gastritis is an exception, for, as we shall see in the next lecture, its symptoms are those which are common to many functional affections—that is to say, they consist chiefly in discomfort rather than real pain after taking food, along, often, with nausea and loss of appetite, but not usually with vomiting, and with but little wasting. The symptoms, then, are comparatively slight, and physical signs of organic disease are absent. On examining the stomach you detect no tenderness, no dilatation, and no tremor, and it is only by the use of the stomach-tube that you can make sure of the diagnosis. But the results yielded by the stomach-tube are conclusive. The most characteristic point is the presence of mucus in excess, so that the contents are more or less viscid and filter badly. In the great majority of cases there is a diminution in the acidity of the contents. Occasionally you

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meet with an excessive acidity (so-called acid-gastritis); but in most cases where there is an excess of mucus the acidity is lowered.

I have told you that gastritis closely simulates functional disorders of the stomach, but the organic affection which it most resembles is carcinoma in its quite early stages. Indeed, I do not think you can with certainty, from the symptoms or from the use of the stomach-tube, tell a case of gastritis from one of early carcinoma, and probably for this reason—that the mere presence of carcinoma sets up a catarrh, so that you do get the signs of gastritis, and you can understand how difficult it may be to distinguish one from the other. I shall refer to that point further when I come to speak of carcinoma.

Chronic Gastric Ulcer.

Chronic ulcer is a typical organic affection, in that all the symptoms of organic disease are markedly present: it is always accompanied by pain, usually by vomiting, and always by more or less wasting. The patient with chronic gastric ulcer may be of either sex, although it is slightly commoner in men, and about the middle period of life. This is the usual kind of history that the patient will give. He will state that for a long time back, usually months, or it may be years, he has had definite dyspeptic symptoms, off and on, better sometimes, worse at others, but pain always predominating. There may have been vomiting, and if so, it has relieved the pain. Further, he will tell you that the pain tends to occur in a definite relation to the taking of food. And in this respect it differs from some of the other kinds of abdominal pain which I considered with you in the first lecture. To some extent the time after taking food before the pain comes on depends upon the situation of the ulcer; the nearer to the pylorus—roughly speaking—the later after a meal will it be felt. You will also be told, very likely, that the degree of pain is largely determined by the kind of food taken, and that solids cause much more than liquids. Sometimes the pain in cases of ulcer is excessive; I have known it so bad that the case has been mistaken for one of biliary colic, the patient lying on the floor and rolling about in agony. The pain is not usually continuous, in which respect it differs from the pain of carcinoma, unless the ulcer has come to involve the serous coat of the stomach, or has perforated, and formed adhesions to the abdominal wall, when it may be constant. As I have already stated, vomiting is frequent, but not invariable, and you must not suppose that hæmatemesis is at all a necessary symptom. It is no more necessary to wait for the onset of hæmatemesis before you diagnose gastric ulcer than it is to refuse to diagnose a case as one of gall-stones because the patient has not had jaundice. If hæmatemesis does occur, however, it is a symptom of great value, but, remember, there are other conditions which may cause a patient to vomit blood besides gastric ulcer. If you have a patient at the time of life I have spoken of, and who gives a history extending over a long period, in which pain has predominated, and if with that pain there has been a certain amount of wasting, you may diagnose ulcer. With regard to the physical signs, they are of comparatively minor importance. I have told you before, and would remind you again, that the physical examination is of less importance in the diagnosis of all these conditions than the history. But the signs which you expect to meet with in a case of ulcer are tenderness over the stomach, which is often confined to a limited area, so that you can put your thumb over it, which is where the ulcer is. It is not a diffuse tenderness such as you find in gastralgia, but a localised spot of tenderness over the ulcer. If the ulcer be so situated as to block the pylorus or induce pyloric spasm, as an ulcer at the pylorus often does, then the stomach will be more or less dilated. But, remember, the ulcer need not be so situated, and that there may be no dilatation of the stomach at all. If the ulcer be near the cardiac end, for instance, there will not be dilatation.

Test-meals are of very little value in the diagnosis of gastric ulcer, and you can safely dispense with their help. Often the result of the test-meal is normal, but if the ulcer be setting up a catarrh the acidity of the contents may be below normal. In the case of an ulcer uncomplicated by catarrh, however, it is usual to find the acidity of the contents rather high. Nor do I consider the X rays of much use in the diagnosis; certainly they are not necessary. You can diagnose cases of gastric ulcer very well without the help of the radiographer, and more

often than not he proves to be a broken reed in cases of doubt. There is one condition, however, in which X rays help you, and that is where what is known as an hour-glass stomach has resulted from an ulcer. But after all there is no practical advantage in diagnosing the existence of an hour-glass stomach; that is a refinement which is unnecessary in practice, and of no value to the patient. It is sufficient for you to diagnose with certainty that the patient has got a chronic ulcer, and that at once places his case in the hands of the surgeon, who can make the more accurate diagnosis at operation.

The things for which you are apt to mistake a chronic ulcer are, in the first place, duodenal ulcer, of which I shall speak immediately. I would only say here that the diagnosis of gastric from duodenal ulcer is often impossible, and really does not matter much, for if you are satisfied that the patient has got an ulcer somewhere, whether it is one inch on the stomach side of the pylorus or one inch beyond it is of no immediate consequence. The diagnosis from carcinoma also may be very difficult. Here you may be inclined to say that the result of the test-meal will keep you right, but it does not; and for this reason—that many cases of simple ulcer, when complicated by catarrh, lose their free hydrochloric acid, and in that way may come to simulate carcinoma. On the other hand, in cases of carcinoma which have started in ulcer free hydrochloric acid may be present. So a test-meal does not necessarily help you much in the diagnosis. Here, too, then, if the history does not keep you right, it is improbable that anything else will. And again, I say it does not much matter, because in some cases the diagnosis is impossible by any method, however refined. Even on the operating table it is sometimes almost impossible. The essential thing, however, is that the patient be brought to the operating table, for that gives him the best chance of cure in either event.

Duodenal Ulcer.

Duodenal ulcer, although not strictly a gastric affection, may be considered with gastric ulcer, because it is pathologically, for practical purposes, the same disease. In regard to it, however, there is a peculiarity which is of some value, and that is, that duodenal ulcer preponderates enormously in men. Why that is has never yet been satisfactorily explained. Gastric ulcer, which is, pathologically, so similar, tends to occur with more nearly equal frequency in the two sexes, whereas though men have not quite a monopoly of duodenal ulcer, they certainly have by far more than their proper share of it. So you will rightly be biassed at the outset, if you are in doubt, by the sex of the patient. Further, duodenal ulcer tends to occur with greatest frequency in people of about the same age-period as gastric ulcer, namely, between thirty and fifty years of age. The history is, in many cases, perfectly clear, and sufficient alone to base a diagnosis upon. It is as follows: The patient will tell you that, often for years back, he has been subject to attacks of indigestion which have been intermittent, that is, they have cleared up and he has been well between them. The attacks last a varying number of weeks at a time, and during them he has pain, often severe, which occurs late after a meal, having the character of "hunger-pain," because it tends to be relieved by the taking of more food. The attacks tend specially to recur at certain parts of the year, namely, in the spring and autumn, and are always worse in cold and damp weather. A spell of hot weather will often cause an attack to disappear. Further, the attacks often cease quite suddenly. A patient will have typical hunger-pain to-day, while to-morrow he will be free from any pain at all. That is an undoubted fact, and it might put you off diagnosing ulcer, because you might argue that it is impossible for this sudden cessation to occur if the patient have duodenal ulcer, seeing that the ulcer cannot be expected to heal in a night. As to that, I would say that, whatever the explanation be—and I am not prepared with any—there is no question that in these cases the patient may have a duodenal ulcer. Over and over again that has been verified at operation; a patient who comes with such a history of sudden cessation of pain will be proved to have a duodenal ulcer. The fact is that we do not yet understand the mechanism of hunger-pain. I shall have something to say about that when I discuss the very vexed question of hyperchlorhydria, but I would simply say now that hunger-pain is not so simple of explanation as some people think. We do not understand how it is produced, and therefore we do not understand why a patient may suddenly cease to have it. Still, I would repeat

that sudden cessation of pain is quite compatible with the presence of an ulcer.

Such is the usual history, and upon a history of hunger-pain, occurring in bouts or spells, spread over several years, even in the entire absence of physical signs, I am convinced you are justified in diagnosing duodenal ulcer. If you get other symptoms, good and well. You will not get vomiting unless the ulcer has led to blocking at the outlet of the stomach. There may be melæna, but you should not wait for the onset of melæna before you diagnose duodenal ulcer; if you do, you will be putting your patient to a grave and unnecessary risk. You must diagnose the ulcer long before the hæmorrhage occurs. Some attach importance to examination of the stools for "occult blood," as it is called, but that, I believe, is in most cases a work of supererogation. You diagnose from the history. What I have said with regard to the search for "occult blood" applies also to X-ray examination. There are persons who profess to be able to diagnose duodenal ulcer with certainty by the use of X rays. I do not believe them. If you cannot diagnose the presence of such an ulcer from the history of the patient, you will not diagnose it by any other means. The history is *the* thing. A test-meal is of some indirect help. Duodenal ulcer is the condition *par excellence* which tends to go with a high total acidity of gastric contents, probably because, being situated beyond the pylorus, it does not set up a secondary gastritis, for such gastritis always tends to bring down the acidity. Gastric ulcer, on the other hand, is always apt to produce a gastritis, which lowers the excess of acidity which would otherwise be present. In a doubtful case, therefore, a high total acidity points to a duodenal rather than a gastric ulcer.

Those are the symptoms of typical or classical duodenal ulcer where it is situated, as 95 per cent. of duodenal ulcers are, in the first part of the duodenum. But sometimes, that is to say in about 5 per cent. of the cases, the ulcer is in the second part of the duodenum, and when that is so the symptoms are by no means so typical, and I believe that in some cases of ulcer in the second part of the duodenum it is impossible to tell, from the patient's description, whether he is the subject of duodenal ulcer or of gall-stones. Over and over again one has known mistakes in one direction or the other, gall-stones being diagnosed for duodenal ulcer, or the reverse, when the ulcer has been situated in the second part of the duodenum. But here, again, you need not vex yourselves too much, or go laboriously diagnosing which it is likely to be. Either condition requires operation, and the surgeon can tell in two minutes, once he is inside the abdomen, which it is, whereas you may waste hours trying, by elaborate methods, to find out which it is and not be sure even then. In all matters of diagnosis you must be practical; medicine is essentially a practical art, not a pastime or a hobby, nor an amusement for people who like laborious trifling. Your object is to get the patient well quickly and safely, and, in my opinion, you cannot get a patient with duodenal ulcer well without operation, nor one with gall-stones either. So if you decide that it is either gall-stones or ulcer, that is all you need do; you can wait for the operation to see which it is. I have said that it is not necessary for there to be any physical signs in duodenal ulcer at all. But sometimes you find, over the upper belly of the right rectus, a little deep tenderness and rigidity. Do not refuse to diagnose duodenal ulcer if that does not happen to be present; the abdomen may be absolutely normal to physical examination, but yet there may be an ulcer.

Carcinoma.

The symptoms and signs of carcinoma of the stomach will, of course, depend upon the position at which the growth is situated. If it be at the cardiac end of the stomach, or at the lower end of the œsophagus, which is the same thing, the symptoms are those of œsophageal obstruction. There is difficulty in swallowing; the patient feels as if his food stuck somewhere, and usually he indicates the lower end of the sternum as the site, and there is marked wasting. Here X rays are of very great help; they will show you the site of the obstruction, and if you find there is such an obstruction coming on fairly abruptly in a more or less elderly person who has previously enjoyed good health, the chances are all in favour of carcinoma. So there is nothing more to be said about it.

When the growth occurs at the pyloric end of the stomach the diagnosis is also easy, because it will show itself first by causing dilatation of the stomach, the symptoms of which I shall speak of

presently. Thirdly, we come to the most difficult cases, those which give most trouble in diagnosis, where the growth starts in the body of the stomach, usually about the lesser curvature, and spreads along it.

First, as to the history in such a case. If you are to believe the surgeons, you would expect the patient to give you a history of preceding dyspepsia, because surgeons have got hold of the heresy that almost all cases of gastric carcinoma start in an ulcer; they will try to prove to you at operations that an ulcer is the starting-point of the carcinoma, and they have got some pathologists to back them up in this. I can only tell you that, in my experience, like that of most physicians, patients who develop carcinoma of the stomach have just as often—indeed, oftener than not—a clean bill of health as regards their preceding digestion. Certainly most of them do not give a history pointing to chronic ulcer, or anything like it. Of course, you can get a history of indigestion from almost anybody; all of you here have probably had indigestion at times; but I am now speaking of a definite history pointing to ulcer. That, I repeat, you do not get in the majority of cases of carcinoma. I think one possible explanation of this surgical-pathological heresy is, what some of the pathologists are now beginning to suggest—that many cases of carcinoma begin as a carcinomatous ulcer, that the growth is an ulcerous one from the outset; it is not a case of carcinoma supervening upon chronic ulcer, but a carcinomatous ulcer spreading into a general carcinoma, and when the surgeon opens into it he sees an ulcer of course. The man who makes the post-mortem also sees the ulcer, but it does not follow that it was a simple ulcer which has become malignant; it may have been a malignant ulcer from the start. I lay stress on this question because I believe it is of diagnostic value, as pointing to carcinoma, if a patient who has previously had good digestion suddenly in later life begins to have symptoms. At first these symptoms will be very vague, and will point to functional rather than to organic mischief; the patient will not complain at the outset of bad pain or of vomiting; he will complain, as one with gastritis does, of a feeling of weight or discomfort after meals, with loss of appetite, particularly—and this is important—for meat, and—also important—he will waste. Just as pain is the characteristic symptom of ulcer, and just as vomiting, as we shall see, is the characteristic symptom of dilatation, so wasting is the characteristic symptom of malignant disease, and it is, perhaps, the most constant of all the symptoms. A patient with carcinoma tends to waste from the outset, before he gets his other symptoms. As the case goes on he will develop more or less pain, which tends to be rather independent of meals, that is to say, it will be constant, although, perhaps, somewhat aggravated by food. In that respect it differs from the pain of ulcer. Sooner or later vomiting sets in. These will be symptoms.

Now as to the signs. In the early stage there are no signs at all. You may examine the abdomen with all the care you please in carcinoma of the body of the stomach, and you will not, at the outset, find any recognisable signs at all. Later on you will begin to feel a tumour. And the tumour is felt in a characteristic situation, namely, between the xiphisternum and the left costal margin, and it can be felt coming down when the patient takes a long breath. It is hard, and sometimes irregular. That may be all in the way of physical signs that you detect on examination. One naturally looks for other diagnostic acids. Do test-meals help? The typical result in a test-meal is the same as that in gastritis, a diminution of acidity with a certain excess of mucus, because, as I have said, carcinoma tends to set up a gastritis. So you see the test-meal does not help you much in distinguishing carcinoma from ordinary gastritis. In those cases in which carcinoma originates in an ulcer, again, there may be normal acidity. So here, also, the test-meal does not help you much. Nor are the X rays of help in the diagnosis in the early stage, unless the growth be at the pylorus, where anyone can diagnose it without the use of X rays. Once a tumour has developed, then you can be pretty sure, but before that event occurs, the signs and symptoms are extremely ambiguous. And by the time a tumour can be felt you are too late. Hence various methods have been invented with a view to diagnosing the condition early, but I shall not trouble you with them, because they have not helped us in the least. There is, unfortunately, it must be confessed, no way of diagnosing, with absolute certainty, a carcinoma in the early stages

of development. You can be more or less suspicious; the man who has applied elaborate tests may be more suspicious than you are who have not tried them, but beyond that you cannot go; you cannot be sure. Hence I think it comes to this: that in a patient at that period of life when carcinoma is common, who develops rather suddenly symptoms pointing to gastritis—symptoms which do not clear up under simple treatment—you are justified in exploring. After all, that is the only certain way of making the diagnosis. I say that because I am convinced that carcinoma of the stomach should be diagnosed earlier than it is; and that if it were diagnosed early enough, the prognosis would be ever so much better than it is. The condition is far more amenable to surgical treatment than people believe, if it can be got early. The mischief is that a patient is allowed to drift on for weeks or months under a diagnosis of gastric catarrh, which is the diagnosis usually made; he is given a stomach mixture, and the fact that he is wasting steadily is not paid attention to, or is attributed simply to the careful dieting he is undergoing. I would advise you, therefore, to pay great attention to the question of wasting; it ought to help you to make the diagnosis from gastric catarrh, because patients with gastritis do not waste in the way that patients with carcinoma do.

Organic Dilatation.

In the last place, to-day, I shall speak of dilatation of the stomach, that is to say, organic dilatation. This leads us, first of all, to the discussion of one or two methods of diagnosis which I did not mention in the first lecture, because, before you can be sure the stomach is dilated you have to be clear as to your methods of determining its size. Fortunately, simple methods are sufficient.

First, try to ascertain, by percussion, the position of the lower border of the stomach as the patient lies on his back. That should be at about the level of the tips of the tenth costal cartilages. But, for convenience, one may say that the greater curvature should not come below the umbilicus. If it is below the umbilicus you may regard the stomach as dilated, provided the lesser curvature is normal in position. You must percuss lightly. I am always emphasising in the wards the importance of light percussion of the abdomen. Mistakes are often made by using too heavy percussion. By that simple method alone, in most cases, you can determine where the greater curvature is. If you are doubtful about the position of the greater curvature, owing to the stomach being empty, you can, if you wish, inflate it by giving the patient a little tartaric acid to swallow (30 gr.), followed by bicarbonate of soda (40 gr.), or you may give a tumblerful of soda-water during effervescence. That will cause enough gas to be liberated to make the results of percussion clearer. You can aid and correct that impression by auscultatory percussion, which you have seen done in the wards, putting the stethoscope below the xiphisternum and scratching the surface along radiating lines, and making a mark where the vibrations set up by the stomach begin. It has been urged in objection to that method that it does not give the exact outline of the stomach, but no one expected it to. What it does give you is the area of the stomach which is in direct contact with the anterior abdominal wall, and if the area of stomach in direct contact with the anterior abdominal wall is greater than it should be, you are usually justified in inferring that the stomach as a whole is dilated. There are some people who refuse to see that, but you will find, in your own experience, that the results of this method of examination are borne out by what you find. You diagnose dilatation by this method, and you find at operations, time after time, that you were right.

Then there is the question of a stomach-splash, If on palpation you find a splash over an area extending below the umbilicus, you may infer the existence of a dilated stomach. But you must be sure the splash is being produced in the stomach and that the latter is not displaced. It is not always easy to differentiate between a splash produced in the transverse colon and one produced in the stomach. Finally, there is the X-ray method, of which I shall say nothing further than that it is very useful where it is available.

So much for determining the existence of dilatation. After you have decided that the stomach is dilated, you must determine whether it is an organic dilatation—which is what I am discussing to-day—or an atonic dilatation. I shall have much to say about atonic dilatation of the stomach at our next meeting. I may say here, however, that organic dilatation is a condition which results from a block at the pylorus, whatever that block may be due to.

In the diagnosis between the two forms of dilatation the history will nearly always keep you right, because in the organic cases there will be a history for years of symptoms of chronic ulcer before the symptoms of dilatation came on; on the other hand, in those cases where the dilatation is the result of a growth at the pylorus, the diagnosis from atonic dilatation should never arise, because no one develops atonic dilatation of the stomach at the period of life when carcinoma develops. Atonic dilatation is a disorder of young persons. The symptoms also will help you, because organic dilatation is characterised by vomiting as its most important symptom. Atonic dilatation is scarcely ever accompanied by vomiting. A patient with even marked atonic dilatation of the stomach very rarely vomits, whereas patients with pyloric obstruction vomit habitually, bringing up enormous quantities of material, which is effervescing, yeasty, and smells sour. So that the history of something leading to pyloric blocking and copious vomiting of that kind, with wasting—because all these patients waste—will, almost alone, make the diagnosis for you. And when you make a physical examination you will be able to tell, by the methods I have described, that the stomach is greatly enlarged, and you will, in most cases—certainly in all advanced cases—be able to see, when the stomach is full, peristaltic waves passing from left to right. When you see those you can be sure, I think, of the existence of pyloric blocking. I would qualify that statement by this, that the only condition I know of in which visible peristalsis may be found without a pyloric block is in people with extremely thin abdominal walls, particularly in women who have borne many children, and in whom the stomach is somewhat displaced downwards. In such cases I have sometimes seen gastric peristalsis when there was no pyloric obstruction. But in the case of the vast majority of patients, if you see stomach peristalsis you are safe in diagnosing the existence of pyloric stenosis. When you have settled that there is a stenosis, it remains for you to say whether it is benign in nature or malignant. In other words, is it the constriction produced by the healing of an old ulcer, or is it due to the development of carcinoma? That point is, usually, easily determined. In the first place, the history will tend to keep you right. The absence of a history pointing to previous ulcer, and the apparently sudden development of the symptoms in a person in the later period of life, who has hitherto had good digestion, will be in favour of carcinoma. A test-meal also helps one a good deal. In benign stenosis you get a very high acidity, with plenty of free hydrochloric acid, and the presence of yeasts and sarcinæ. In malignant stenosis you find a lowered acidity, with no free hydrochloric acid, along with the presence of lactic acid and Oppler-Boas bacilli. But here, again, if you cannot with certainty tell malignant from benign stenosis, you need not vex yourself, because the case is one for the surgeon anyhow.

November 24th, 1913.

ELECTRO-THERAPEUTICS FOR PRACTITIONERS. By FRANCIS HOWARD HUMPHRIS, M.D., F.R.C.P., Fellow of the Royal Society of Medicine. Pp. 243. London: Edward Arnold. 8*s.* 6*d.* net.

The object of this book is to indicate the disease in which electro-therapy will be most generally useful. A descriptive account is given of static electricity, X rays, incandescent light and electro-thermic penetration. A correct and comprehensive definition of pain has been a desideratum. Dr. Humphris thus defines it: "(1) All pain can be traced to pressure even as all pain-relieving agents tend to relieve pressure; (2) our nerves are in a constant state of vibration, and pressure alters the normal rate of vibration; (3) our nerves are conductors of electric currents; (4) with the altered vibration rate, due to the pressure, there is an altered electrical resistance; (5) in the altered electrical resistance the currents pass differently; (6) this difference is pain." Pain, therefore, is defined as the subjective manifestation in a nerve of altered electrical resistance primarily caused by pressure. Chapters are devoted to the treatment of obesity, of carcinoma and sarcoma, lumbago, skin disease, while the last chapter is a homily on lessons from failures. Whether one agrees with the author's views or not the book is most readable and suggestive.

PURPURA.

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On August 18th, 1913, I saw in consultation with Dr. E. D. Evans, of Wrexham, a young lady, æt. 23 years, who was suffering from purpura. When eleven years of age she had a bad attack of purpura, but from then till the beginning of the present attack she has been free. She had never had rheumatic fever, and on the whole had been a healthy girl.

The present attack began soon after last Christmas, when the first thing complained of was blindness in her right eye; this was due to hæmorrhage into the retina. This slowly cleared up, and when I saw her she was able to count fingers five feet away. About a month ago the left eye became affected in the same way, but not so severely. There are also some sub-conjunctival bleedings.

She had attacks of very severe abdominal pain, and also frequently attacks of vomiting, mostly bilious in character. There was a good deal of abdominal tenderness, most marked over the descending colon, but the whole abdomen was more or less tender. When I saw her she was passing blood by her rectum, and she also had hæmaturia. Before the onset of hæmaturia the urine was albuminous. The spleen could not be felt and there was no enlargement of the liver. The lungs were normal, but the heart was dilated, and there was a faint hæmic bruit at the pulmonary area, but there was no mitral murmur.

The purpura came out in crops, and when I saw her the largest spot present was about the size of a florin on her right leg. She got pains in her arms and legs, which tended to be worse in the evening. There was no redness or swelling of any joint when I saw her, though the right knee had been swollen, very tender and reddish the day previously. There was very slight œdema over the dorsum of her right foot. At times she has epistaxis and bleeding from her gums. Her temperature has never been much raised, not above 100° F. Her blood contained 4,000,000 red cells and 80 per cent. hæmoglobin, so that the colour index was 1; there were 19,600 white corpuscles, the increased number being due entirely to the polymorphonuclear leucocytes. This blood change was evidently due to the repeated hæmorrhages. She had had these attacks on and off for eight months, being sometimes well enough to get up and go out, at others confined to her bed.

I saw her again with Dr. Evans on September 3rd; she had been improving since I last saw her up to two days previously, when she became very ill again. There was now dulness over the base of the right lung, and patches of bronchial breathing over both bases. Her temperature was only 99·5° F. The melæna and hæmaturia had both stopped, but there was albumen in the urine.

Purpura is defined as spontaneous extravasations of blood into the skin, mucous membranes, and internal organs, with or without free hæmorrhage from the mucous membranes. It is really a symptom, and occurs in association with many diseases, also as a result of mechanical interference with the circulation. In addition to these causes it is also met with as a distinct entity, for which no cause is known.

Purpura may be conveniently divided into symptomatic, and idiopathic or arthritic. The former is found in association with—

(1) Infectious diseases such as smallpox, scarlet fever, typhus, measles and infective endocarditis.

(2) Nervous complaints. Purpuric spots are seen in some cases of tabes.

(3) Cachectic conditions, as in carcinoma, tuberculosis, and Bright's disease. Under this heading may be placed the purpuric eruptions seen in the blood diseases, pernicious anæmia and leucocythæmia.

(4) The taking of some drugs. Potassium iodide at times produces a severe purpura. This appears to be due to an idiosyncrasy towards the potassium salt, as many people in whom this salt causes purpura can take the sodium or ammonium salt with impunity. A fatal case has been recorded in a child as the result of a very small dose. So well is this recognised that a special variety of purpura has been described as iodic purpura. Phosphorus, mercury and quinine may rarely cause purpuric spots.

(5) Mechanical interference with the circulation. This is well seen in whooping-cough, where sometimes the whole of the conjunctiva is discoloured with effused blood.

At times purpura is met with in a patient in whom none of the foregoing causes are at work; it is then known as idiopathic or arthritic purpura from the fact that there is always more or less joint pain at the same time. Arthritic purpura may be divided into purpura simplex, purpura hæmorrhagica, purpura rheumatica, and Henoch's purpura.

Purpura simplex is a slight complaint; it begins with a little constitutional disturbance, sore throat and pains in the joints, generally the ankles. The eruption quickly appears, and consists of dull red spots varying in size from a pin's point to a quarter or half an inch in diameter. They are not raised and do not disappear on pressure. They fade in two or three days, leaving a brownish-yellow stain. They come out in crops so that on the same patient may be seen several of different colour, some fresh red, others faded yellow. It appears mostly around the ankles and on the lower extremities; rarely is it found on the trunk and upper limb. Recovery takes place.

Purpura hæmorrhagica is an extremely bad name for the next variety as all purpura is hæmorrhagic, and a better name is morbus maculosus, or morbus maculosus Werlhofii after the German physician who first described it in 1775. It is a severe and dangerous form of purpura. It is ushered in by headache, gastric pain, and often vomiting. The eruption is more marked than in purpura simplex, the extravasations of blood being larger, and occurring under the mucous membranes and often under the conjunctiva. It is frequently seen on the penis and scrotum, and bleeding often takes place into the tongue; at times it is so severe that sloughing takes place. When severe in the tongue and throat it may cause great danger to life. In addition to the purpuric staining hæmorrhagic œdema occurs, the areas being red, raised above the surface and pitting on pressure. Hæmorrhage may take place from any mucous membrane, causing epistaxis, hæmoptysis, hæmatemesis or bleeding from the genito-urinary tract. Occasionally a fatal result happens from a hæmorrhage into the brain.

There is pain in the joints and limbs, and frequently albumen in the urine apart from hæmaturia. The temperature is high, and may reach 104° F., or even go higher.

A case with high fever, much extravasation of blood, frequently repeated and severe hæmorrhages from the mucous membranes, may end fatally in a few days (purpura fulminans, or malignant purpura). In a case which is going to terminate favourably the disease may run its course fitfully for weeks, finally leaving the patient weak, anæmic and wasted.

The next variety, purpura rheumatica, Schönlein's disease, or peliosis rheumatica, would be better transferred to the group of symptomatic purpura, as it is always associated with rheumatic fever; but inasmuch as the rheumatic fever element is generally so slight during an attack, it is usual to describe it with the arthritic group.

It occurs between the ages of eleven and forty, and is about equally common in both sexes. It begins with a dull, aching pain in the legs, the pain often being described as a "sense of bursting"; at the same time there is an eruption of bright red spots on the legs, slightly raised, and not disappearing on pressure. The knees and ankles are painful, swollen and tender, and the skin over them is sometimes reddened. Slight œdema of the dorsum of the foot is generally present. These symptoms come out in the late afternoon or evening, and by next morning the pain, swelling and redness have gone, and the bright red spots have become dull red or purple. The second day, or later, there is a return of the symptoms in the evening.

The purpuric spots pass through the usual changes in colour, so that on the same patient there may be seen bright red raised spots from a millet-seed to a threepenny-piece in size, dull red or purple spots of the same size (none of them disappearing on pressure), and also yellowish-brown stains. The symptoms are symmetrical, both ankles or both knees being affected, and in the first attack are usually confined to the area below the knees; subsequent attacks may involve the thighs and upper extremities, but very rarely is any purpura seen on the trunk or face. Hæmorrhages from the mucous membrane are very rare, but occasionally a few spots may be seen on the buccal mucous membrane.

The temperature is only slightly raised, and unless the joints are very bad there is no sweating. The patient can walk fairly well in the morning, but in the evening walking is difficult, or impossible, owing to stiffness and pain. Exertion tends to bring on an attack. It may persist for weeks, but is a mild complaint, and is but rarely fatal.

The evidence of its connection with rheumatic fever is very clear; sometimes rheumatic fever is diagnosed before the purpura appears, and nearly all the cases have a history of either rheumatic fever, tonsillitis, or chorea, or there is evidence of old endocarditis.

The last variety, Henoch's purpura, is characterised by the association of marked abdominal symptoms, such as vomiting, colic, and intestinal hæmorrhage, with purpura and arthritic pain and swelling.

It may begin with pain and swelling of the joints, followed by purpura, colic, vomiting, and melæna; or gastro-intestinal derangements may precede purpura, articular swelling and pain. The illness consists of a series of such events extending over a long period of weeks or months. Recurrence is one of its marked features, and it may extend over several years.

The temperature is very slightly raised, or there may be a complete absence of pyrexia. The eruption is of the usual purpuric type, and is mostly found on the extremities, but it is seen fairly frequently on the face and trunk. Most frequently it is purely hæmorrhagic, though occasionally hæmorrhagic œdema is seen. The colic is usually very severe, and quite distinct from it there is a marked abdominal tenderness on palpation, most noticeable over the colon. The vomiting is severe and at times most difficult to control; it is generally bilious in nature, but sometimes hæmatemesis occurs. The motions at some time or another during an attack contain blood. The urine is nearly always albuminous, and not infrequently hæmaturia occurs as well. Epistaxis and hæmoptysis may occur as well.

During an attack, one day one joint, another day a different joint becomes painful and stiff, and the skin over it may be reddened. The pain and stiffness do not last perhaps more than a few hours. There may even be an effusion of fluid into the joint. The dorsal surfaces of the feet are frequently swollen.

There is no doubt, I think, that the case I saw with Dr. Evans is one of Henoch's purpura. It certainly is not purpura simplex, and it differs from purpura hæmorrhagica in that there has been practically no pyrexia, whereas in purpura hæmorrhagica the temperature is high. Again, in our case the rash has never been anything but a simple purpura, and has been seen on the face round the eyes, and on the trunk. In hæmorrhagic purpura the rash is rarely seen anywhere except on the extremities, and hæmorrhagic œdema is generally present with the simple purpuric rash.

It differed from purpura rheumatica in several respects. There was no history of rheumatism or allied complaints, there was no "sense of bursting" pain when the eruption appeared. Most marked was the difference in the absence of hæmorrhage from mucous membranes in Schönlein's disease, while in this case bleeding took place from practically every mucous surface. In purpura rheumatica also the symptoms are usually symmetrical, while in this case there was absolutely no suggestion of symmetry.

The diagnosis of any case of purpura will in the first place have to be made between symptomatic and idiopathic purpura. This as a rule will not cause much trouble except in the case of the blood diseases and scurvy. An examination of the blood will at once show if the case be one of pernicious anæmia or leucocythæmia. If the case be one of scurvy, the peculiar characteristic is the sponginess of the gums: at first sight the clotted blood round the teeth in a case of purpura might be mistaken for this sponginess, but washing the mouth out will prevent this mistake. Again, in scurvy the hæmorrhages are mainly subcutaneous and intra-muscular, causing hard brawny swellings in the calf muscles, or they occur under the periosteum over the tibia, where they produce swellings not unlike gummata.

On the whole the prognosis is good. Purpura simplex always recovers, unless it is the early stage of purpura hæmorrhagica. The older the patient the worse the prognosis becomes. The gravity of a case is as a rule seen early, and depends on the severity and frequency of the hæmorrhages, whether the mucous membranes are bleeding or not, the degree of pyrexia, the amount of blood destruction, and the albuminuria.

The treatment consists of rest in bed, with nourishing liquid food and stimulants if necessary, and if the purpura is associated with some other complaint, the treatment appropriate for that disease. For drugs, calcium chloride in 10- or 15-gr. doses every four hours, or the lactate. Adrenalin in doses of from five to thirty minims or combined with 3- or 4-minim doses of Fowler's solution every four hours does good at times. Probably the best drug of all is turpentine in doses of ten or twenty minims in a capsule or suspended in a mixture thrice daily. For the resulting anæmia iron should be given. In Henoch's purpura an ice-bag to the abdomen is often of service.

November 24th, 1913.

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PSORIASIS.*

By H. G. ADAMSON, M.D., F.R.C.P.,

Physician for Diseases of the Skin at St. Bartholomew's Hospital.

PSORIASIS is one of the more common affections of the skin. It is an eruption which has very characteristic features, little liable to modifications from secondary changes; so that it is generally easy to recognise. It does not as a rule affect the general health, and as there are usually no symptoms of itching or discomfort from discharges, a patient will not always seek advice unless the uncovered parts are affected. Although psoriasis has characteristic features which mark it out as a distinct and definite disease, due probably to some one particular cause, we are quite in the dark as to what is this cause. The most striking ætiological fact is that the complaint often runs in families, but this gives us no clue to its origin. There is no evidence that it is an infectious disease.

The eruption consists of sharply circumscribed, red, scaly patches. It tends to be distributed widely and symmetrically, and may involve any part of the skin, although it has a preference for certain areas. These favourite situations are the extensor surfaces of the limbs, especially the elbows and knees and the scalp. It may sometimes avoid these parts altogether, but in doubtful cases the finding of patches upon the tips of the elbows or knees is often of great help in diagnosis. Other situations in which psoriasis may occur are the palms and soles, the nails, and also on parts which are, as a rule, left free, namely, the flexures. The face may be attacked, but commonly is not, unless on the forehead just at the margin of the scalp. The sharply circumscribed patches of which the eruption is made up are sometimes bright red,

* A lecture delivered at the Hospital.

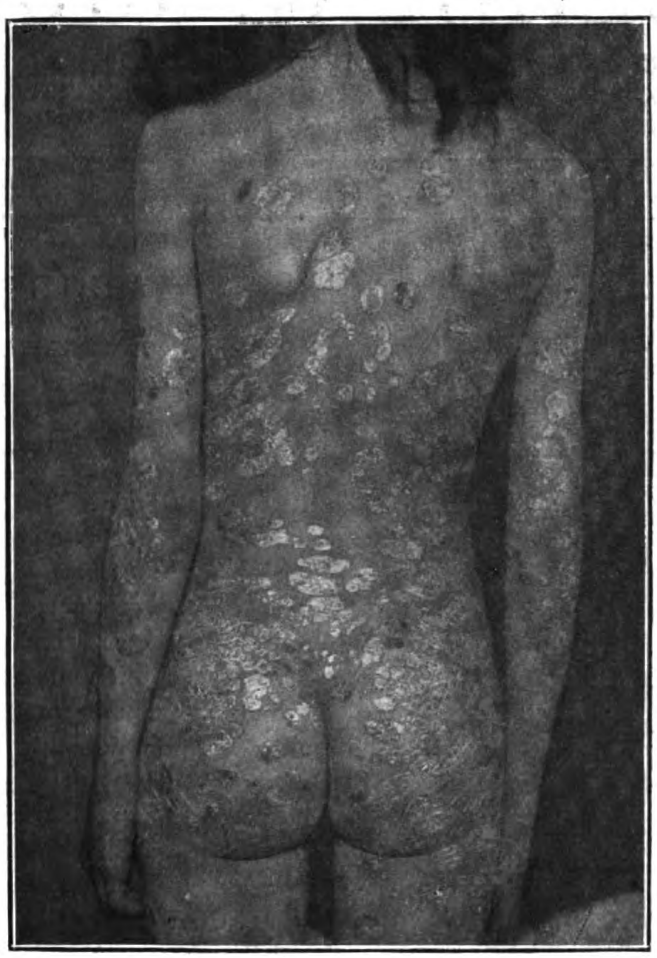

FIG. 1.—Psoriasis vulgaris. The eruption consists of sharply circumscribed rounded patches which are covered with silvery or mica-like scales. On the back some patches have spread out into broken rings (psoriasis circinata).

more often dark red, and are covered with silvery or mica-like scales. These silvery scales form one of the most striking features of the psoriasis eruption. It is important to remember, in regard to the scaling, that this may be masked by ointments, or may not be evident in quite recent lesions, but that if the patch is scratched with the finger-nail, the scales at once begin to appear and to flake off. And if one meets with an eruption of very sharply circumscribed patches, one instinctively scrapes a patch to see whether silvery scales will appear. Another thing to note about these patches is that they are not papules; for there is no thickening of the skin from cell-infiltration. The patches are little more than slightly vascular areas covered with scales—points to which I shall refer again when I consider their histology. The sizes of the patches may vary greatly from that of a pin's head up to large areas several inches across. They are primarily rounded (Fig. 1); but irregular shapes may be formed by the coalescence of two or more patches. Patches sometimes tend to fade in the centre, and spread as rings; and when these coalesce gyrate figures are formed (Fig. 2). Indeed, it is almost entirely from differences in size of the lesions, in their degree of scaliness, in their distribution, or in chronicity of the patches that different appearances are produced in different cases. Names are given to describe these various types, such as psoriasis punctata for the very small lesions; psoriasis guttata and psoriasis nummularis for more usual size patches; psoriasis discoides, psoriasis circinata, psoriasis gyrata, psoriasis inveterata (Fig. 3), psoriasis rupioides—when the scales are much heaped up, etc.

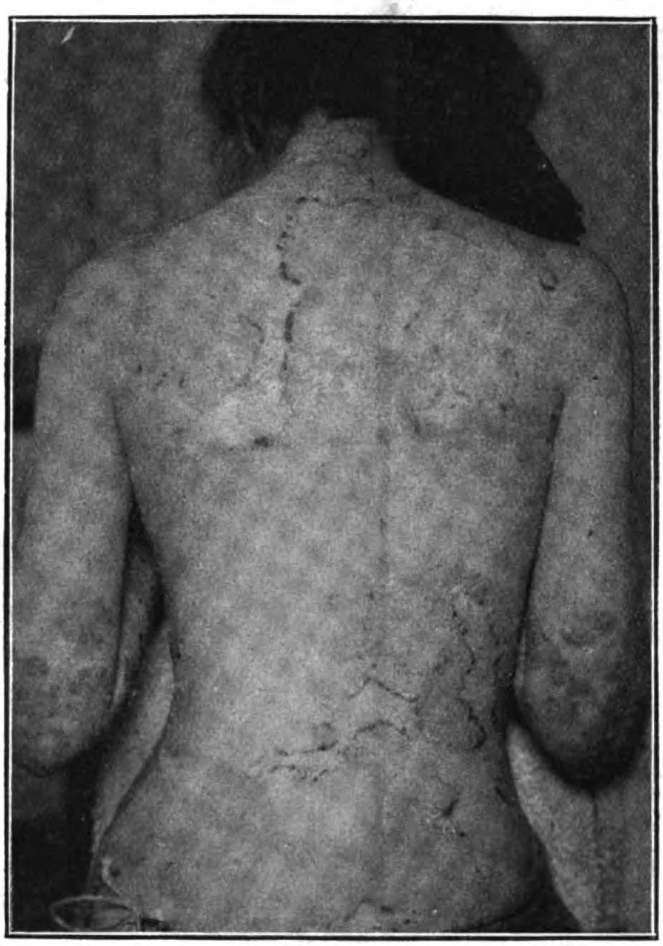

FIG. 2.—Psoriasis gyrata. The original patches have extended into rings, which have coalesced to form gyrate figures.

One other point to which attention must be called is that every patch is made by extension from a centre, and not, as are the patches of some eruptions, by coalescence of small lesions. This may seem a small matter, but it is of importance in distinguishing psoriasis from Lichen planus, in which the patches can be seen to be made up by coalescence of smaller papules.

Psoriasis in special positions.—I have already said that psoriasis occurs in the scalp—indeed, it is seldom absent from the scalp when present elsewhere. On the scalp-patches the scales are generally much thicker and more heaped up, and the redness of the skin is less marked. Their patchiness distinguishes the scaling from that of seborrhœa capitis, and the fact that the hairs do not fall out or break off from ringworm. Sometimes the scalp patches may creep over just on to the forehead or neck in the form of scaly rings or festoons.

The *nails* are affected in a certain proportion of psoriasis cases. They may be covered with little shallow pittings, like the dents on a thimble (Fig. 4). This is common, and sometimes a help in the diagnosis of psoriasis. Or there may be scaly patches on the bed of the nail separating the nail-plate at one part (Fig. 5). This latter form of nail psoriasis has to be distinguished from ringworm of the nails, and the distinction may be difficult when, as sometimes happens, no other psoriasis patches are present.

The *palms* may sometimes be involved in a general eruption of psoriasis in the form of multiple small scaly patches (Fig. 6). Or they may be affected in large scaly areas, and then often with little or no eruption on the body (Fig. 7), so that

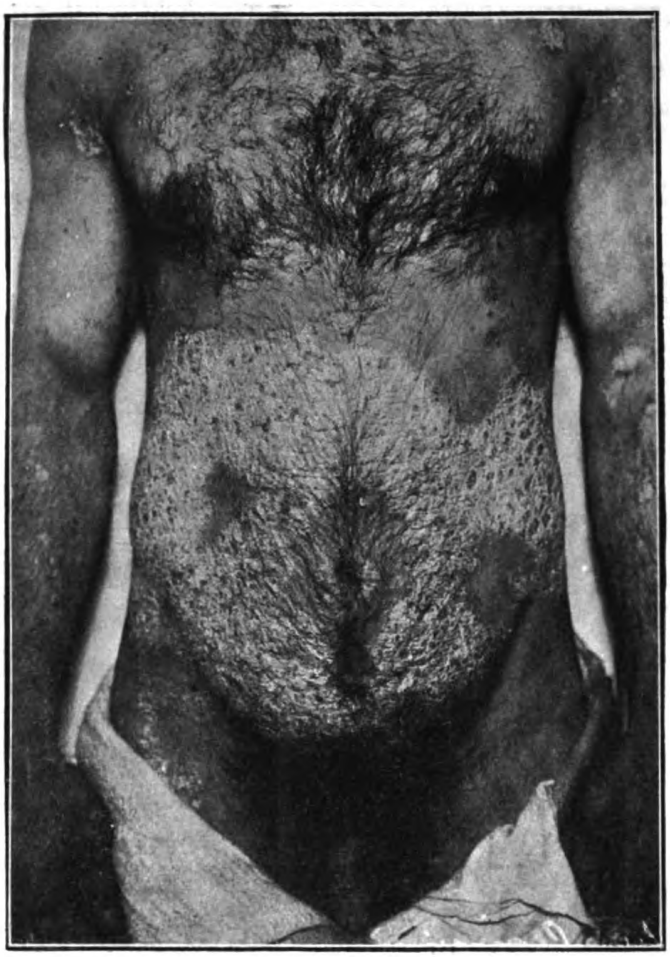

FIG. 3.—Psoriasis inveterata. Shows very large patches which were of long standing, but, except for the heaping-up of scales, having the same characters as more recent patches.

diagnosis may be difficult from other scaly affections of the palm, such as eczema and palmar syphilides.

Flexural psoriasis.—It is a curious thing that psoriasis, which generally avoids the flexures, may sometimes occur there, and be almost, if not quite, limited to those parts. Commonly the groins, or the axillæ, or the spaces between the toes may be involved. When occurring in the flexures it is very likely to be mistaken for seborrhœic dermatitis.

Symptoms.—Rarely a patient with psoriasis may complain of itching; but as a rule there is no itching, and no other subjective symptom in psoriasis—only the discomfort, and if on the face or hands, the disfigurement of the eruption.

The course of psoriasis.—The course taken by the disease varies much in different cases. The first attack may occur at any time of life, from infancy to old age; but once attacked, the subject of the disease is very liable to future outbreaks. This is one of the outstanding features of psoriasis—the tendency to recurrence throughout the patient's life. A patient—a child or an adult—once having had psoriasis may be afterwards periodically attacked, especially in the spring or in the autumn. Or the interval of freedom may be longer—perhaps several years, or even many years. The extent and duration of the eruption also varies in different patients, and in different attacks in the same patient. It may be an extensive punctate eruption, an ordinary guttate or nummular type all over the trunk, or it may be confined to certain parts—the elbows and knees and scalp—or it may even attack the nails and palms alone. In

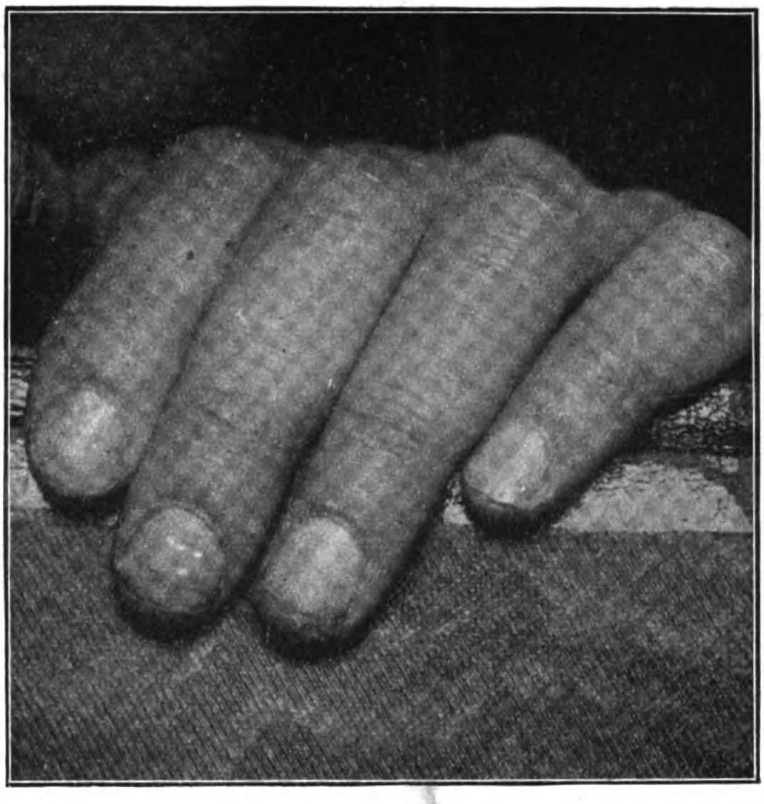

FIG. 4.—Pitting of the nails in a case of psoriasis. A common feature which is often of help in diagnosis.

FIG. 5.—Psoriasis of the nails. Scaly patches have formed on the nail-beds. The nails over the patches have become opaque and toughened, and are separated from their beds.

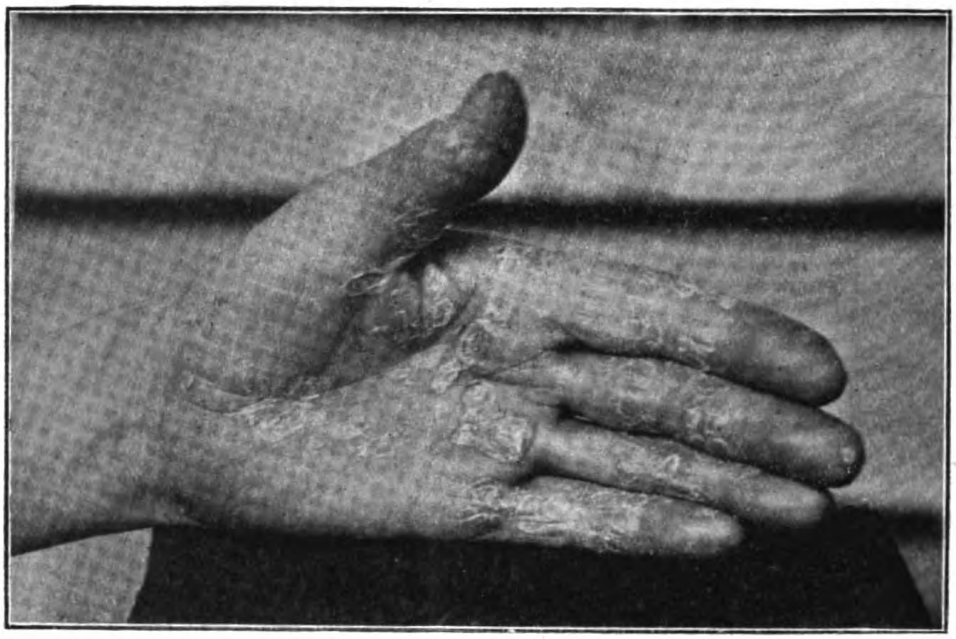

FIG. 6.—Psoriasis palmaris. Distinguished from secondary syphilide by the absence of coppery colour and by the uniformly silvery character of the scales on scraping.

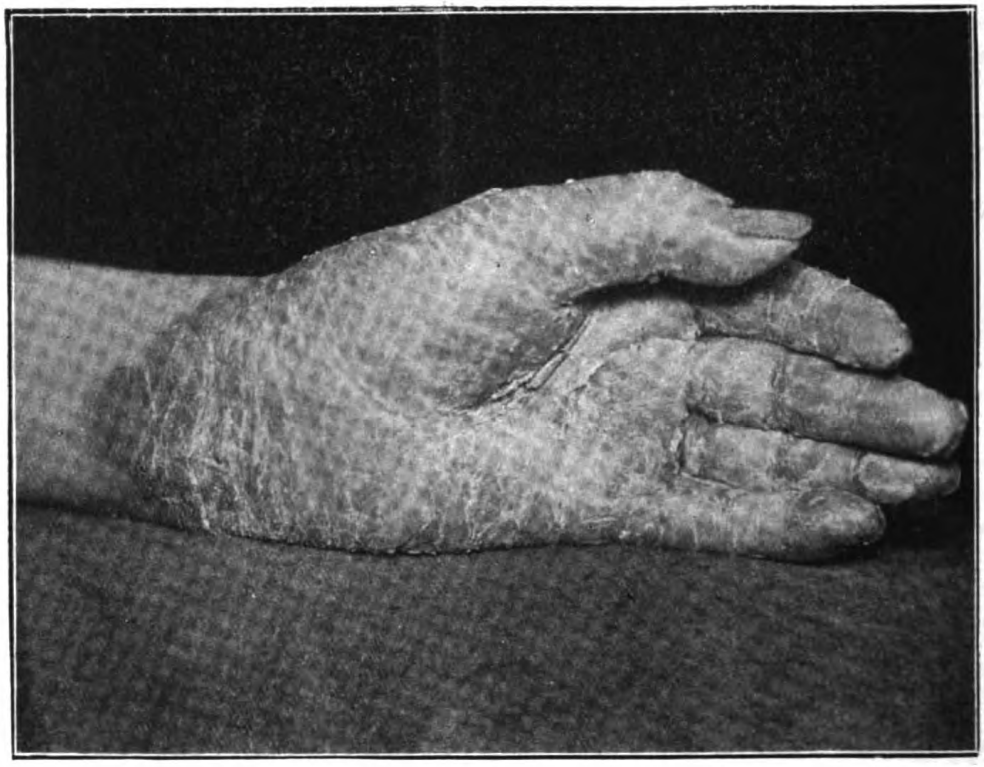

FIG. 7.—Psoriasis palmaris. The palm of the right hand and both soles were similarly affected, but there was no psoriasis elsewhere. During a subsequent attack the limbs were affected in the usual way, but the hands remained free.

rare cases it may be so extensive that hardly a clear space of skin remains. Sometimes the eruption may persist for years, and in some of these long-standing cases very large patches may be formed.

Ætiology and pathology.—The causation of such a common and well-defined disease as psoriasis has been naturally much discussed. But the cause has not yet been discovered. The view most usually held is that it is a parasitic infection, but no micro-organism is found in the lesions, and there is little, if any, evidence of contagion. The one striking feature in its ætiology is the fact that it is distinctly a family disease. Sometimes many members of a family for several generations are found to have been troubled with this complaint. Sometimes there is also asthma in the family. Occasionally, especially in elderly people, psoriasis is associated with rheumatoid arthritis. But persons with psoriasis are nearly always otherwise in good health. It has been noted that many persons attacked by psoriasis have red hair. The pathological anatomy of the lesions is in many respects unlike that of any other eruption, and although it does not help to suggest the origin of the eruption, it explains very well the clinical appearances. Microscopically there is found:

(1) A dilatation of the vessels of the superficial plexus and of the papillary vessels, with a very moderate amount of cell-exudation round the vessels.

(2) Evidence of active growth of the prickle-cell layer with a dipping down of the epidermis in long processes between the enlarged and vascular papillæ.

(3) An imperfect cornification of the horny cells, due probably to too active proliferation and to the imperfect formation into horn-cells owing to there being some œdema of the prickle-cell layer.

(4) Between the layer of horny cells are seen collections of round-cells which have come to the surface from the dilated vessels beneath the epidermis.

From this microscopical examination we see why the patches are red and scaly and also why they do not feel infiltrated. But it also explains something further, namely, the value of the "scraping test," a test which is often most useful in diagnosing psoriasis from syphilis. If a patch of psoriasis be scraped with a blunt instrument at first there form more silvery scales, but after further scraping all the scales are removed and then there is reached a smooth, red, dry, glazed surface. Pressure with the finger on this red glazed surface makes it white; there is no infiltration and the blood is pressed out of it. Further scraping of the surface produces a number of minute bleeding points, these points being the apices of the vascular papillæ. These events only happen with psoriasis, and if we scrape a psoriasis-like patch which we think may be syphilis, we can soon tell the difference, because a syphilitic papule is made up of a dense cell-infiltration in the skin, and if we continue to scrape it, instead of reaching a smooth, glazed surface with bleeding points, we find ourselves digging out the tissue.

I have dwelt upon this apparently simple matter because it is really of great importance as a help in the diagnosis of a "psoriasiform syphilide" from psoriasis.

Diagnosis.—The features of a psoriasis eruption are so characteristic that one can seldom make a mistake. The sharply circumscribed patches with silvery scales; the tendency to wide distribution, with preference for extensor surfaces and elbows and knees, and scalp; the absence of symptoms and good general health; the tendency to recurrence, and the fact that often other members of the family are affected, are all characteristic features.

A common mistake is to diagnose psoriasis as a *syphilide*; but generally a little attention to a few points should enable one to avoid this mistake. It is true that the secondary eruptions of syphilis are often wide-spread, sharply circumscribed and even scaly, like psoriasis; but psoriasis patches differ in that they are not infiltrated like syphilitic eruptions. It is sufficient in a doubtful case to apply the scraping test just described. Other evidences of syphilis are often present—remains of a primary sore, sore throat, loss of hair, gland enlargement, mucous tubercles, anæmia, etc.—but even without these, careful examination of the lesions should enable one to make a diagnosis.

Lichen planus may sometimes be mistaken for psoriasis, especially when the papules of lichen planus run together into patches, which may become a little scaly. I have already said that the patches in lichen planus can be seen to be

made up of a grouping of papules. Further, in lichen planus there is intense itching; the primary lesions—the angular, flat-topped, dusky-red papules —are seen if carefully looked for; the eruption has certain favourite sites—about the wrists, on the back of the neck, round the waist and inside the thighs and knees, and, unlike psoriasis, it may occur on mucous membranes.

Another eruption which may sometimes be mistaken for psoriasis is *Pityriasis rosea*. This is a common eruption, but not a well-known one. It is sometimes mistaken for psoriasis, sometimes for syphilis. It begins as one patch—the herald patch—then, after about ten days, it bursts out over the trunk and upper part of the limbs in many round or oval, pinkish, slightly scaly patches, which often itch much and which generally fade away after a few weeks. Pityriasis rosea is possibly an infectious eruption, for it often comes in small epidemics.

Psoriasis of the flexures should always be thought of in the presence of obstinate scaly, sharply circumscribed red patches in the groins, or axillæ, or between the toes. Other affections which may occur in these regions are seborrhœic dermatitis and ringworm. Seborrhœic dermatitis is associated with seborrhœa capitis, and is easily cured with sulphur ointment, which at once distinguishes it from psoriasis of the flexures, which sulphur does not affect. Ringworm of the groins or axilla or between the toes is less scaly than psoriasis, and the fungus is found on microscopical examination of the scales after mounting in liquor potassæ.

Psoriasis of the palms is often very difficult to distinguish from syphilis or eczema of the palms, and it is sometimes possible to make a diagnosis only when there is psoriasis elsewhere, or eczema elsewhere, or other signs of syphilis. The same may be said of psoriasis of the nail, with the addition that ringworm of the nail has also to be thought of.

Prognosis.—One can generally promise to cure any eruption present, but one cannot prevent further outbreaks, although I believe that if one can get rid of every scrap of eruption present a recurrence may sometimes be warded off for many years. At any rate one can always assure the patient that the affection is a skin disease and that it will not affect the general health.

Treatment.

There is no doubt that internal medication may sometimes exercise a powerful curative effect on psoriasis; but the results obtained by the use of drugs given internally are always uncertain and less rapid than those obtained by external medication alone.

Internal medicaments operate either by their influence on the nutrition of the skin or by inducing alterations in the general nutrition. Their curative action depends upon the intensity of the physiological changes to which they lead, and it is always necessary to push such remedies to the extreme point of tolerance. For this reason they are better avoided, and only out of respect for tradition is it necessary to study some of these internal methods. The drugs which have been most commonly employed are arsenic, iodide of potassium, thyroid extract, and salicin or salicylates.

Arsenic.—In the employment of arsenic in psoriasis the drug must be pushed to its full extent of tolerance, and its curative action is not felt until it has been administered for several weeks. Although in some cases it may cause the eruption to disappear, its action is unreliable, and more frequently it has no good effect. Its use does not prevent recurrences. The most convenient method of administration is as Fowler's solution. Beginning with three minims (in water) three times a day after meals, the dose is increased by one-minim gradations until a dose of ten minims is reached, always provided the patient exhibits no gastro-intestinal symptoms. Having reached this dose it is again dropped gradually to three minims, and again advanced to ten, as before, and so on. No patient should be put on a long course of arsenic without being carefully watched.

Iodide of potassium has been strongly recommended for psoriasis. But its action is also uncertain, and it must be given in very large doses.

Thyroid extract has had also many advocates. It undoubtedly cures many cases, but it also more frequently fails. The drawbacks to its use are well known, and it should not be given unless the patient can be kept under observation in bed.

Salicin or salicylates have been advocated,

especially in early or spreading eruptions. The dose must be a large one—from 15 to 40 gr., three times a day. Its use has no serious inconveniences, and it is sometimes worth trying in cases where one especially desires to avoid ointments, although the choice may be given to the patient between the less irksome, but uncertain internal remedy, and the more disagreeable but certain external method of treatment.

External remedies.—There is probably no case of psoriasis which cannot be cured by external remedies properly and perseveringly applied. The method of treatment adopted must depend to some extent on the case. In a recent case it is better to use some mild and clean application such as an ointment containing acid salicylic 15 gr., hyd. ammon. chlor. 20 gr. to the ounce of vaseline or lard; to be rubbed in after a warm bath. The same may be said of a case in which the attacks are known to recur and to last only a few weeks. But in cases where the eruption has lasted perhaps for years, it is worth the patient's while to undertake a more unpleasant but more effectual treatment by tar or by chrysarobin. Tar is a remedy which has for long been employed, and which is still largely used. It may be prescribed in various forms and combinations. The following are simple and useful prescriptions:

℞ Ol. cadini . . . . ʒ j – ʒss.
Vaselin ad . . . . ʒj

℞ Picis carbonis præparatæ . ʒj – ʒss.
Vaselin ad . . . . ʒ j

℞ Ol. cadini vel picis carb. præp. ʒij – ʒss.
Saponem mollem ad. . . ʒ j

℞ Liq. picis carbonis, B.P.

Before the use of an ointment the scales should be removed so far as is possible by soaping and by soaking in a warm bath. The tar and soap mixture is best employed just before the bath. Both ointments and soap mixture must be well rubbed in. When tar applications are being used over large areas the urine should be watched, and if any darkening occurs, indicating toxæmia, which is rare, the application must be stopped.

One of the best of these tar applications, especially if the patient cannot remain in bed during the treatment, is the liq. picis carbonis B.P. This is scrubbed into every patch once a day with a short, stiff brush. It soon dries, and has not therefore the disadvantage of an ointment which greases and stains the clothes.

Although with many, tar is the favourite remedy for psoriasis, another drug—chrysarobin—which is also commonly employed, is much more efficacious and more rapid in its action. Its use is attended by certain drawbacks. It stains the linen, and the stains do not wash out. If it gets into the eyes it causes a violent and painful conjunctivitis, and it is also liable to produce acute erythema at the thinner parts of the skin, such as at the flexures of the joints. These evil effects can be avoided by clothing the patient in old garments, by forbidding the application of the remedy to the scalp, face, and hands, and, except with care, to the flexures. It is generally better to keep a patient in bed during the chrysarobin treatment. Baths are best avoided until the termination of the course of treatment. The simplest way of applying chrysarobin is in an ointment with vaseline. We may begin with gr. x to the ounce, and increase to ʒj to the ounce for obstinate patches. The effect of the chrysarobin application is to stain the skin around the patches, while the patches themselves become less scaly and less red, and finally appear as pale areas in the stained and reddened skin around. When they reach this condition the application may be discontinued and a bath may then be given. For the scalp and hands, ointments containing tar, or the following—naphthol, 20 gr.; resorcin, 20 gr.; vaseline, ʒj, which is more cleanly and odourless—may be used. For the scalp a preliminary shampoo, using Hebra's spiritus saponis alkalinus as a soap, removes the scales and favours the application of ointment.

For chronic patches of psoriasis when limited in number and extent, X-ray applications are very useful. The best method of procedure is to give three applications of one third of the depilation dose (one third Sabouraud pastille dose) at intervals of ten days, *i. e.* a full pastille dose is spread over a period of one month.

December 1st, 1913.

LECTURES
ON
THE DIFFERENTIAL DIAGNOSIS OF CHRONIC GASTRIC DISORDERS.*

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LECTURE IV.

FUNCTIONAL DISORDERS.

GENTLEMEN,—Functional disorders of the stomach, as opposed to organic disease of that organ, are, as we have already seen, characterised by the fact that they cause discomfort rather than real pain and little or no wasting, and that in them vomiting is comparatively infrequent. These are the three main distinguishing features of the functional as opposed to the organic affections.

Any of the functions of the stomach, secretory, motor or sensory, may be disordered, and so give rise to symptoms of dyspepsia.

We shall consider disorders of secretion first.

In order to prove the existence of disordered secretion the administration of a test-meal is essential. You cannot with certainty, from symptoms alone, tell what sort of secretion a patient has got. I shall not now go into the technique of the administration of test-meals, because you will be able to find that in your text-books, but I repeat that if you wish to make sure of the nature of the gastric contents you must use the stomach-tube. You will remember that, taking the results of the ordinary test breakfast, the total acidity of the stomach contents is estimated in terms of decinormal soda. The number of c.cs. of decinormal soda required to neutralise 100 c.c. of gastric contents is the measure of the total acidity, and taking that basis, you find that the normal is about from 30 to 70 c.c. of decinormal soda (orthochlorhydria). When the secretion is disordered it may be either above or below that. A rise above 70 is spoken of as *hypersecretion* or *hyperchlorhydria*, the meaning of which term we shall discuss immediately, and it may be anything up even to 100, although that is very rare; an acidity of 90 is very high. On the other hand, it may fall below 30, and anything between 10 and 30 is spoken of as *hypochlorhydria*. If it falls below 10 it is called *achylia*.

EXCESS OF SECRETION.

The terms "hyperchlorhydria" and "hypersecretion" are both used in respect of cases where there is excess of acidity. Strictly speaking, by hyperchlorhydria one means a condition in which the percentage of hydrochloric acid in the stomach is above the normal. By hypersecretion one means cases in which the percentage of hydrochloric acid is normal, but the total gastric juice secreted is in excess. So the difference is that in hyperchlorhydria the stomach is secreting a stronger juice than normal, one in which there is more acid, but is not secreting more in total quantity. In hypersecretion it is secreting juice which is more abundant, but not stronger than it should be. There are some who deny that there is any such distinction. They say that all cases of "hyperchlorhydria" are really cases in which there is a hypersecretion, that the stomach never yields too strong a juice, and any excess is in the direction of a too abundant secretion. That is rather an academic question, and I shall not trouble you just now with any discussion as to which view is correct, because I do not think that, for clinical purposes, it much manners. Practically we have to consider cases in which acidity is in excess, and if you want a special word to designate them you may speak of them as cases of hyperchlorhydria, whether there is too high a total acidity or whether it is in the percentage of HCl. which is increased. It must be confessed, however, that the word "hyperchlorhydria" is a rather unfortunate one. It is one of those terms which is, I think, in process of disappearing from medicine, perhaps with advantage. By "hyperchlorhydria" we used always to mean this kind of case: A patient, usually a young man, otherwise in perfectly good health, complains of discomfort, sometimes amounting to pain, in the stomach region, coming on in the late periods of digestion, two and a half to three hours after a meal. This pain is relieved by taking more food; it is immediately relieved also by taking an alkali. These symptoms, without any others—wasting, vomiting or anything

* Delivered at the London Hospital.

else—recurring at intervals of a few weeks at a time, and then disappearing again, make up the condition which used to be spoken of as hyperchlorhydria, and which is not so very uncommon in young and otherwise healthy men; probably several of you have suffered from it at one time or another. The question is, Is this really a purely functional condition? Is it a condition which can exist without any organic lesion? That has recently come to be doubted. Certainly the results of surgical operations on the abdomen have shown that in a large number of cases presenting these symptoms, and with a test-meal showing over-secretion, there is some organic basis for the trouble. Perhaps the commonest organic lesion is duodenal ulcer, which, as we saw at the last lecture, is accompanied by a very high degree of acidity.

Another organic lesion which may cause hyperacidity is chronic appendicitis; you sometimes find it also in a patient who is the subject of gall-stones. But even allowing for all those cases, I think there is reason to suppose that sometimes this condition which we call hyperchlorhydria is really a purely functional disorder. We have to admit that it is so only in a minority of cases, but I do not think we are able to put aside altogether the possibility of purely functional cases occurring. I say that because there are cases on record—I know of at least two myself—in which, in the first attack of such indigestion, operation has been performed and no organic lesion found, though all the internal organs, including the duodenum, appendix and gall bladder, were examined. Therefore I say I think we have reason to believe that, although it may be exceptional, still in some cases at least of what used to be described as hyperchlorhydria, the condition is, so far as we can determine, one in which there is no gross organic cause. It is very probable that if a patient goes on having such recurring attacks of hyperacidity, he will develop a duodenal ulcer, and that, I think, is what happens if the condition is not cured. But, fortunately, in the majority of cases the patient, after one or two attacks of hunger-pain, ceases to have them, and nothing further develops.

The way in which the pain is produced in cases of hyperacidity is very difficult of explanation. The natural explanation advanced is, of course, that the high degree of acidity irritates the stomach and so produces pain. In favour of that view is the fact that if you give a patient who suffers from this type of pain a full dose of bicarbonate of soda he immediately loses the pain. Hence it is said that the pain is due to the acidity. But there must be something more, because it has been found—and you can make the experiment on yourselves—that one can swallow a considerable quantity of dilute hydrochloric acid, even if it has an acidity well above 100, without experiencing any pain at all. Therefore one is forced to the conclusion that mere hyperacidity of gastric contents, in itself, does not cause pain directly, and you must either assume that in addition to the excess of acid there is over-sensibility or hyperæsthesia of the stomach, or that the pain is produced by the acid causing a violent contraction or spasm of the pyloric end of the stomach. The acidity alone will not explain it; it is a factor, of course, because if it were not, the pain would not be immediately relieved by giving alkali. As a matter of fact—and this is of clinical importance,—when a patient gets symptoms as a result of hyperacidity alone, he will be found to complain of epigastric discomfort and acid risings into the throat, popularly called heart-burn, rather than of hunger-pain. Therefore, clinically, you will be more inclined to suspect over-secretion as the sole cause of the patient's troubles when he complains of burning sensations after meals. If he complains of dull aching pain, which is relieved by alkalies, and relieved by taking food, you will be more inclined to suspect some organic basis for the condition. That is all I shall say about over-secretion of acid; and I would only impress upon you again that, in the majority of cases, I believe it has an organic basis, and in only comparatively few cases is it a purely functional neurosis.

Defect of Secretion.

Now to pass to the cases in which the gastric juice is in defect (hypochlorhydria and achylia). Mere deficiency of gastric juice does not seem to produce much in the way of symptoms at all, so long as the motor functions are normal. Where the total acidity falls below 10 you are more apt to get symptoms. But even achylia is an extraordinarily silent condition. A patient may have it for years without having anything which he would speak of as real indigestion. Curiously

enough—and this is a most deceptive thing—patients who have achylia, with almost a total absence of gastric juice, often complain of acidity. The reason they complain in this way seems to be that the absence of gastric juice allows of the production of organic acids—such as lactic and acetic—by fermentation, and it is these organic acids which produce the symptoms. Be not deceived, therefore, in the matter of achylia by the fact that the patient complains of "acidity." Far more often, however, the symptoms in a patient with achylia are referable, not to the stomach, but to the intestine, and manifest themselves in a chronic and recurring diarrhœa. The tendency to protracted or recurring diarrhœa is one of the commonest ways in which the absence, or even marked deficiency, of gastric juice shows itself. It is not of very uncommon occurrence. I have seen this week two patients who suffered long from intractable diarrhœa, in neither of whom were there any particular stomach symptoms, yet in both there was achylia. The conditions from which you must diagnose achylia are gastritis, or chronic catarrh of the stomach, and malignant disease, both of which are accompanied by great diminution of secretion, sometimes almost by complete absence of it. I have told you how to diagnose gastritis and carcinoma, so I shall say nothing more about that, but I would only raise here, just as I did in the case of over-secretion, the question whether achylia really ever is purely functional. It depends to some extent on what you mean by "functional" and "organic." If you mean can the stomach cease to produce gastric juice without showing, *to the naked eye*, any sign of gross disease, it certainly may. But it is possible, and perhaps even probable, that in all those cases there are microscopic changes in the mucous membrane, an atrophic condition which has led to the destruction of the secreting-cells. This is certainly true in some cases of achylia, and it may be true in all. Certainly, as far as my own experience goes, I have never known a case of achylia pass off and the secretion become re-established. I know such cases have been recorded by others, but they must be extremely rare. I think that once achylia has become established it always remains, and that looks as if one were dealing with an organic condition. But remember there is no gross change, nothing that surgery can deal with; and if the lesion be organic at all it is of a microscopic character.

Disorders of Motor Functions.

I now pass from disorders of secretion to consider what is a more easy subject to handle—namely, disorders of the motor functions of the stomach. The motor functions of the stomach are of two kinds, which it is well to keep separate. There is, first, the *peristaltic movement* of the stomach, by which it expels its contents; and there is, secondly, *tonicity*. Theoretically, both these might vary, either in the direction of excess or of defect. We know that exaggerated peristalsis occurs, for instance, in cases of obstruction at the outlet of the pylorus. When we were discussing dilatation of the stomach we saw, you will remember, that one of its signs was the occurrence of visible waves of powerful peristalsis sweeping across the stomach. I think it is doubtful, however, whether you ever get exaggerated peristalsis by itself and without obstruction. Certainly if you do it does not produce symptoms which one can recognise. Nor, apparently, does a defective peristalsis alone tend to occur often. It occurs along with deficiency of tone, as you will see later. There again, if it does occur, it is difficult to attribute symptoms to it. You will always be tempted to speak of "pyloric spasm," by which, I suppose, is meant exaggerated contraction at the pyloric end of the stomach, as an explanation of all sorts of abdominal pains. I warn you particularly against making a diagnosis of pyloric spasm; it lies in wait like a pit for the unwary; it is so easy to try to satisfy the mind of the patient, if not your own mind, by explaining any violent epigastric pain as a pyloric spasm or "cramp at the outlet of the stomach." The vast majority of cases diagnosed as pyloric spasm have an organic basis. They are either cases of gall-stones or of ulcer situated near the pylorus, leading, I quite admit, reflexly, to spasm; but it is the ulcer which is the important thing. But I do not think you ought to believe in the existence of functional pyloric spasm. I do not believe it occurs.

Disorders of Tonicity.

Alterations in the tone of the stomach may be in the direction either of deficiency or of excess. Deficiency of tone is one of the commonest causes

of dyspepsia. A large number of functional dyspeptics—most of the people, in fact, who have what is popularly called a "weak stomach," the habitual life-long dyspeptics—are people who have deficient gastric tone, acquired, or in some cases inherited, for it seems sometimes to be a family peculiarity. It is extremely important that you should have clear notions on what is meant by "gastric tone." Gastric tone may be defined as the power of the stomach to grasp its contents when the vertical posture is assumed by the individual. Tone comes into play apparently by reflex action so soon as the vertical position is entered upon, and the consequence of that is that, no matter how much material you introduce into the stomach, the stomach, by virtue of its tone, is able to accommodate itself to its contents, so that the food always remains at the same level. The stomach, in fact, contracts round the food in the form of a tube, the width of that tube being proportional to the quantity of food introduced. There are some people in whom the tone reflex is defective, and in whom this adaptation of the stomach to its contents does not take place. Such people are said to have atonic stomachs, and they get symptoms which I shall describe immediately. A liability to atonic dyspepsia may thus be regarded as one of the payments which we make for the doubtful privilege of walking about in the upright posture. The atonic stomach, instead of holding its contents up, sags down, and the contents drop into the lowest part. It behaves, in fact, like a loose bag. If the process goes on such a patient may finally have a truly dilated stomach, in which some of the contents come to lie at a lower level than the pyloric orifice. Such a stomach has difficulty in emptying itself, and in such a case you may get some retention of contents.

Now, with regard to the symptoms of which patients with defective tonicity complain. They complain not of pain, but of a sense of fulness and discomfort after meals, and of sinking and emptiness, an "all-gone, faint sensation," as they will tell you, in the epigastrium when the stomach is empty. These patients hardly ever give a history of vomiting, although if the atony has gone on to dilatation, vomiting may occasionally occur. Nor do these patients waste; they are almost always naturally spare and ill-nourished, but progressive wasting does not occur. On physical examination patients with gastric atony can often be distinguished by even a superficial examination; they are usually people of a congenitally neurasthenic or delicate build. They often have that narrow costal angle of which I spoke in the first lecture. It is people with that type of abdomen who are particularly liable to have atony of the stomach. They are, in nearly every case, under-nourished, and they remain under-nourished in spite of eating generously—a paradox which I cannot go into just now, and which is not yet fully understood. On local examination you will find more or less tendency to dilatation of the stomach; it seems to be a little larger than it should be, although not necessarily reaching below the umbilicus; in particular you find that the fundus extends unusually high into the axillary region, often reaching the level of the sixth rib in the mid-axillary line. Further, these patients always exhibit a splashy abdomen; sometimes they will notice the splash themselves, and it can always be elicited by palpation, remembering the precautions to which I drew your attention in a previous lecture.

Gastroptosis.

One may conveniently refer here to the condition called gastroptosis, because gastroptosis very often goes with atonicity. Indeed, gastroptosis alone, unless there be also want of tone, produces little in the way of symptoms. By gastroptosis one means a dislocation or dropping down of the stomach as a whole, so that, in extreme cases, it may come to lie in a horizontal position across the centre of the abdomen—the so-called "hammock" or "sling" stomach. More commonly the dropping down affects the pylorus, so that the stomach assumes a more vertical position than normal. But, whatever the exact form of the dropping, it tends to be accompanied by want of tone. Gastroptosis is easily recognised, if you are on the look-out for it, in most cases. It is far commonest in women, although men are not by any means immune, and it occurs usually in association with dropping of other organs, being part of a general visceroptosis, which affects the colon, the pelvic organs in women, and the kidneys. You can frequently recognise gastroptosis at once on examination of the abdomen by the fact that there is, in the upper part, an unusual hollowness, with an unusual fulness lower down as the patient lies on the back. When you

come to percuss out the stomach in the usual way, you find that the lesser curvature has fallen down, and that the greater curvature is below the umbilicus, but that the distance between the greater and lesser curvature is not increased, that is to say, it does not exceed four inches. That is characteristic of ptosis of the stomach as a whole, as distinct from dilatation of it, because in a dilated stomach the lesser curvature remains under the liver and the greater curvature goes down, and the distance between the two curvatures is more than four inches. In the lesser degrees of gastroptosis you may have difficulty in determining the condition, because when the patient lies on the back the stomach tends to go up again. The lesser degrees can be made out most easily, just as can the lesser degrees of atony by the use of the X rays.

Gastralgia.

I now pass to a consideration of the sensory disorders of the stomach. Here again, theoretically, sensory functions may be increased or diminished. But in this case you are met at the outset with this difficulty—that the normal mucous membrane of the stomach is *insensitive*. How, then, can you increase or diminish a thing which does not exist? That, of course, is rather a poser. We shall get at it best in this way: Increased sensibility of the stomach—I am not now discussing whether it is right or wrong—according to the clinical view, occurs, and to it the term "gastralgia" is applied. And the clinician will tell you that this condition of increased sensibility of the stomach tends to occur almost exclusively in young women; that it is characterised by the fact that the patient gets pain almost immediately food enters the stomach, whether the food be liquid or solid, and that the pain may be so great as even to lead to vomiting, so simulating, to some extent, organic disease, although it does not usually lead to wasting. Further, if you examine such a patient you will find that the whole stomach area is tender on pressure; the whole epigastrium is tender; there is not merely one tender spot, such as one finds in gastric ulcer.

Now, there can be no doubt that such a type of dyspepsia as this is extremely common in young women; you will see it in the out-patient room every day. And the question arises again, just as it arose *à propos* of achylia and hyperchlorhydria, Is it really purely functional? Is there no lesion of the mucous membrane? If you examine such a stomach, take it out and open it up and inspect it, will you find nothing abnormal? Is it purely a case of the nerves being too sensitive? I think we must admit that in such cases a purely functional condition, mere hypersensitiveness and nothing else, does not exist; that in all those cases, if you could examine the mucous membrane, you would find it altered in some way—probably congested, or even perhaps inflamed. There is a tendency for little erosions to form, and for the stomach, at the same time, to produce too much secretion. And we know that in some of those cases there is hæmatemesis. Now, hæmatemesis, as a result of a purely functional condition, is impossible; you cannot imagine a stomach which bleeds without a lesion in the mucous membrane, no matter how small. Hence it is that the term "gastralgia" is not a satisfactory one, though it is a convenient clinical term to apply to the group of cases above described, and in which over-sensitiveness of the mucous membrane may be part of the pathology. The process is also apt to go on to the formation of erosions, or even small ulcers, which lead to bleeding, and when it does that, you may speak of it as "gastro-staxis" or "hæmorrhagic gastralgia."

I warned you, you will remember, against making a diagnosis of "pyloric spasm," and similarly, although you may speak to your patients about "neuralgia of the stomach" if you like, do not deceive yourselves with any such expression. Most so-called neuralgias of the stomach are something else—gall-stones, or true ulcers. Reserve, if you like, the term "gastralgia" for the particular group of cases in young women, such as I have described, but realise that you are dealing with an organic condition of microscopic dimensions, it is true, not a gross surgical change, but an organic one all the same, and just as the so-called hyperchlorhydria seems to predispose patients later on to duodenal ulcer, so I think "gastralgia" in young women seems to predispose them in later life to true gastric ulcer.

If over-sensitiveness of the stomach as a cause of symptoms be so doubtful, what shall we say of under-sensitiveness? We know of no condition which may be due to a still further depression of the gastric sensibility, for, if there be normally no sensibility, you cannot depress it; nothing from

nothing is nothing, therefore there is nothing which corresponds to gastralgia in the negative direction. Some have suggested that hysterical anorexia—a profound want of appetite met with in hysterical patients—is due to a depression of the sensory functions of the stomach, but when you get into that domain you are in the region of pure speculation, and one person's guess is as good as that of another.

In closing, gentlemen, I would say that in connection with diseases of the stomach, just as in the whole sphere of pathology, you will find the same thing—the organic steadily encroaching on the functional. Every advance in knowledge tends to show that conditions which we have been in the habit of speaking of as functional have an organic basis. Therefore, perhaps I should qualify what I have said so often throughout these lectures, that organic conditions of the stomach are for the surgeon to deal with, by saying *gross* organic states are for the surgeon to deal with; but that amongst the medical or functional conditions, so-called, there are several in which there is really a microscopic-organic change at the bottom of the trouble—inflammatory, congestive or other. Still, even though the organic is steadily encroaching on the functional in pathology, I would have you remember that the majority of cases of dyspepsia which you will meet with in practice are to be called functional, a large proportion of them being due to atonicity, combined, often, with perversions of secretion in one direction or another. So that, though it may come about in the future that there are more varieties of organic disease of the stomach recognised, yet the functional will still form the majority of your patients.

December 1st, 1913.

Recovery from Tuberculous Meningitis.—Reichmann and Rauch report two cases of recovery from meningitis with tubercle bacilli in the cerebrospinal fluid; one patient was a child less than two years old, the other a robust young man. Both were treated by lumbar puncture and the application of a Bier constricting band. They quote 18 other cases of recovery. The child had lumbar puncture done 5 times, withdrawing a total of 55 c.c. of fluid. The fluid was under 500 mm. water pressure and it kept high in spite of the improvement, being 300 mm. at the time of the last puncture.—*Journ. A.M.A.*, vol. lxi, p. 443.

DOES HERPES ZOSTER EVER OCCUR TRAUMATICALLY?

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By the publication in 1900 of his elaborate investigations into the pathology of herpes zoster (1), Head established a thesis which has met with practically universal acceptance, viz. that zoster is essentially due to a lesion—largely hæmorrhagic in nature—of the posterior root-ganglion in the affected area. His later article in 'Allbutt's System of Medicine' (2), dealing with the same disease, gives his more recent views as to ætiology, incidence, etc., from a clinical standpoint. In neither of these communications, however, is there a definite pronouncement as to whether herpes zoster can be produced traumatically. In his earlier article, no doubt, a case is described (3) in which a slight attack of herpes in the left ninth intercostal space appeared twelve days after the patient had sustained a fracture of the eleventh dorsal vertebra from a blow on the back by a descending lift. It is evident, however, from the microscopical report upon the ninth posterior root-ganglion, that the lesions typical of herpes zoster were not pronounced, *e.g.* "there is no sign of a hæmorrhage." Trauma, of course, is mentioned by several writers among the possible exciting causes of zoster, and cases of herpes following an injury are common enough, but are these not rather *post* than *propter hoc* instances? Crocker (4) says: "Zoster has occurred in several instances as the result of contusion or other lesions of the terminal nerve-filaments, such as on the cheek and eyelids following a blow," and goes on further to state that "zoster is produced by any irritative lesion or condition in any part of the tract from the cord to the periphery of the nerve supplying the affected skin." The "proofs of this," which he goes on to give, are based very largely upon cases and investigations reported prior to the date of Head's published work, and do not appear altogether convincing. Moreover, if it is generally accepted that "*any* irritative lesion or condition of *any* part of the tract" can "produce" herpes, it is rather difficult to see why Head's investigations regarding root-ganglia should have

been so widely recognised as of much import. It should be borne in mind, also, that in his later contribution Head states that "crops of bullous eruptions are described as occurring after injury to peripheral nerves," but goes on to say, "it is doubtful if such eruptions should be classed with zoster" for reasons stated. Cases associated with spinal caries have, of course, been described, but what I have hitherto been unable to find is a definite statement as to whether an attack of herpes can be produced by "an accident." The idea of "shingles" being due to an accident may appear improbable and I make no attempt here to offer proof on the matter, but rather ask the question, Is it possible for hæmorrhage into a posterior root-ganglion with more or less secondary degeneration to be brought about by trauma of nerve-roots?

The query presented itself in a concrete form in the case of a patient whom I had to see with reference to the question of his claiming under the Workmen's Compensation Act or not. Briefly the notes of the case are these:

Case 1.—F. F—, æt. 46 years, seaman, was admitted to Leith Hospital on September 4th, 1909, with the following history: Five days previously, while his vessel was in the Thames, he was sent in a small boat to fasten a rope to a buoy. While in the act of making fast the rope, standing in the boat with his back to the steamer, the rope suddenly tightened (through the vessel swinging), giving him a violent blow on his right side below the armpit, nearly knocking him into the water. In his attempt to save himself, his right arm was violently twisted and wrenched as he was whirled round, clinging still to the moored rope's end. On the voyage to Leith he suffered considerable pain below his armpit, and on visiting the hospital out-patient department his symptoms were naturally ascribed to muscular bruising, no eruption being then visible. On his return, however, two days later, the real nature of his condition was evident and he was at once admitted.

He exhibited on his right side the most extensive and the most "confluent" eruption of herpes zoster I ever saw, covering nearly the third, fourth, fifth and sixth dorsal segmental areas (as mapped out in Head's diagrams). Partly, no doubt, as the result of the bruising of the skin from the blow, the erythematous base was of a markedly livid hue, and over a considerable area of the eruption suppuration occurred, so that it was nearly five weeks before he could be discharged.

Now the question I had to deal with was whether his incapacity "arose out of an accident," and it was not without some hesitation that I certified that in all likelihood it did. I do not profess an intimate enough acquaintance with anatomy to prove the facts, but it appeared to me that it might be possible, by a forcible tearing of the scapular attachments plus a severe twisting of the trunk, so to stretch nerve-roots as to cause hæmorrhage into the root-ganglion. That he had herpes zoster none of my colleagues who saw the case had any doubt.

Another case, seen since then, was also suggestive of traumatic origin.

Case 2.—J. E—, æt. 28 years, millwright. On April 9th, 1912, while assisting to carry a heavy weight, he fell through a hole in the floor of a loft, his left armpit catching on the edge of the opening and thus suspending him. For a few days he suffered from stiffness, but on the 16th burning pains came on in his left scapular region, and when seen on the 20th he showed distinct herpetic spots behind at the level of the fifth and sixth spaces on the left side and below the left axilla in the corresponding area.

Now, in view of the working of the National Insurance Act, it is of no little importance for practitioners to have some authoritative opinion as to whether such cases as these—and other similar cases must have occurred—are to be certified as suffering from "sickness" (drawing "sick pay" under the Insurance Act) or from "accident."

It is not without interest, in the light of the present outcry about "malingering," to note that, as regards F. F—, the captain in *his* report expressed the view that the man *had brought on the eruption so as to escape duty*—a new instance of dermatitis artefacta!

References.

(1) 'Brain,' vol. xxxiii (1900), pp. 353 *sqq.*

(2) Clifford Allbutt's 'System' (1910), vol. vii, pp. 470 *sqq.*

(3) 'Brain,' *ibid.*, p. 483.

(4) 'Diseases of the Skin' (1905), vol. i, pp. 235 *sqq.*

December 1st, 1913.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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FLEXION OF RIGHT HIP-JOINT.

By LAWRIE McGAVIN, F.R.C.S.Eng.,

Surgeon to the Dreadnought Hospital and to the Hospital for Women; Lecturer on Clinical Surgery in the London (Post-Graduate) School of Clinical Medicine.

GENTLEMEN,—The boy you see before you is æt. 14 years, and has been admitted for flexion of right hip and inability to fully extend his right thigh. His history is as follows: Up till four days ago he was apparently in perfect health, never having suffered from any illness nor having received any injury. On getting up four mornings ago he noticed that he was unable to straighten his thigh, and that on attempting to do so it caused him some ill-defined pain which he referred to the iliac fossa. He therefore remained at home, being unable to walk without discomfort, but as during the last three days the flexion and pain have rather increased, he was brought to hospital yesterday, and his condition, as you will note if you examine him carefully, is as follows:

He walks in a curious doubled-up attitude, inclining the pelvis to the right, flexing the hip and resting the right hand on the thigh. Consequently he has to point the toes in order to reach the ground. His attitude is thus somewhat like that of an old faulty ankylosis of the hip-joint. Placed in bed he lies on his back with the left hip extended and the right flexed to an angle of about 145 degrees: on this side there is neither internal nor external rotation. In appearance he is well nourished and of a good healthy colour, with broad shoulders, wide costal angle and good chest expansion. His muscles are well developed; his tongue is clean and he has every indication of being in excellent health. His temperature, however, varies from 100·5° to 102° F., and has done so for the past forty-eight hours. On manual examination you will

FIG. 1.—Attitude on admission. Front view.

notice that the right thigh can be flexed almost completely on to the abdomen without causing him any appreciable discomfort; as soon, however, as any attempt is made to extend it he assumes the position of lordosis, the lumbar spine being thrown forwards, and he at once complains of pain in the region of the iliac fossa. In the position of flexion internal and external rotation are almost normal, although the former is hardly so free as the latter. These movements give rise to no pain nor to any sensation of grating in the joint. There is no swelling over the joint, no wasting of the thigh muscles, and there is a complete absence of tenderness when the head of the femur is pressed into the acetabulum from the trochanter, or through the long axis of the bone. Adduction is painless, but on abducting the limb pain is at once complained of in the region of the groin and iliac fossa. Lastly, his knee-jerks and plantar reflexes are normal.

If you turn your attention to the iliac fossa you will find that there is nothing abnormal to be felt: there is neither rigidity of muscles, tenderness nor

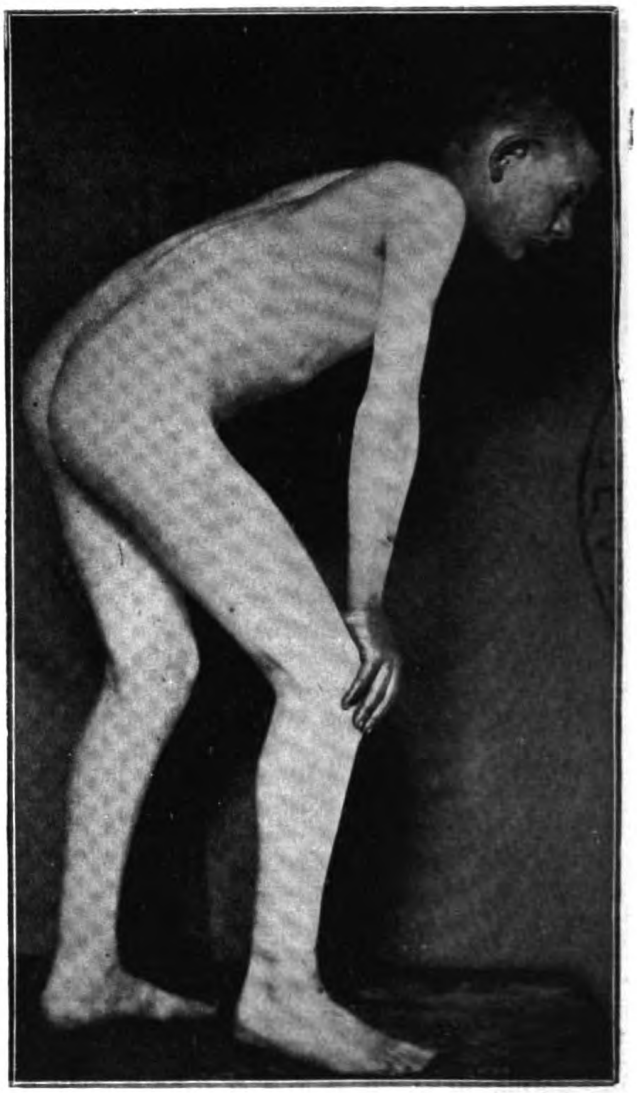

FIG. 2.—Attitude of patient on admission. Side view.

swelling to help you. Now, gentlemen, the first questions I would ask you to put to yourselves are these: Is the flexion of the hip the result of involuntary muscular spasm? Is it a voluntary action on the part of the patient to relieve some pathological condition causing tension? Is it due to some condition causing permanent shortening of muscles or ligaments? Or is it due to some change in the hip-joint itself?

In other words is it due to (1) hysterical contracture; (2) tubercular disease of the hip-joint; or (3) irritation of the ilio-psoas muscle or lumbar plexus?

In the first place the patient is not of the type in whom you would expect any manifestation of neurosis, and he is hardly at the age for such. He has so far given no indication of any such affection; the onset was sudden, and the condition is accompanied by pyrexia. No distraction of his attention makes any difference to the limitation of movement, and his expression is characteristic of genuine pain on attempting to force these movements. The occurrence of lordosis on extension is unusual in patients unless they have active disease or are clever enough to know the significance of this sign when malingering. Secondly, the position of this thigh does not suggest hip-joint disease, since, as you are aware, that disease in its earlier stage results in the position of flexion, abduction and eversion, and in the later stages flexion, adduction and inversion. This patient exhibits flexion alone, and there is little difficulty in everting or inverting the thigh provided this flexion is maintained. The history is far too short for a diagnosis of tubercle of the hip-joint, and his symptoms are not nearly acute enough to warrant one of septic arthritis. If we could for a moment admit the possibility of an acute effusion into this joint, two signs would still be lacking, viz. the eversion always seen when the joint is placed in the position which will accommodate the greatest amount of fluid with the least possible tension, and the presence of swelling and fulness in the groin pointing to distension of the joint capsule. The absolute painlessness of any movement except extension, and the absence of tenderness on pressing the joint surfaces together are entirely against the existence of any joint disease. What, then, are the conditions which, in the absence of hysteria and joint disease, will produce flexion at the hip-joint? Cicatricial contractures of the skin and fasciæ, as from burns, suppurations, etc., need not be considered here, since none are present; therefore only such conditions as will bring about shortening, either temporary or permanent, of the muscles governing flexion of the joint remain to be considered. These muscles are the iliacus, the psoas, and the pectineus. The approximation of the origin of any or all of these muscles to their insertion must result in flexion of the hip-joint, and in the case of the latter muscle a certain degree of external rotation and adduction. For this reason, as well as that this muscle is entirely extra-pelvic in its origin and is rarely involved in any pathological condition, it may well be excluded from consideration. Thus we have narrowed the question down to conditions causing shortening of the iliacus and psoas muscles. Now consider for a moment how such shortening may be brought about. It may be the result of nerve irritation, muscular inflammation, or disease of bone. For all practical purposes the iliacus and psoas muscles may be looked upon as one—the ilio-psoas muscle. Their nerve supply is derived from the upper portion of the lumbar plexus through the second and third lumbar nerves, the fibres being given off for the iliacus about the centre of its inner border and for the psoas during the passage of the nerves through its substance. Thus an irritative lesion affecting these nerves may be situated between the middle of the body of the ninth dorsal and the lower margin of the eleventh dorsal vertebra. Such a lesion might be found in an osteo-sarcoma of the spine or a neuroma (fibro-sarcoma) of the nerves themselves. Such nerve irritation would result, in the earlier stages, in tonic contraction of the muscles involved, but would be of very gradual onset; it would ultimately give way to secondary paresis if not relieved by operation.

On the other hand, primary inflammatory lesions originating in the ilio-psoas muscle are of the rarest occurrence—indeed, by some authorities their existence is denied; but secondary inflammations are met with, and of these the commonest is, of course, tuberculosis associated with caries of the spine, sacro-iliac disease, or more rarely acetabular caries. Local irritation or even destruction of muscle may arise as the result of a perinephric abscess, or an inflamed appendix may become adherent to the muscle-sheaths and result in an intra-muscular abscess. All of these conditions

will be accompanied by pain, tenderness, flexion of the hip-joint and pyrexia. It thus becomes your business to consider which of these conditions is at the root of the trouble in this particular case.

Let us take the most commonly met with first, viz. a psoas abscess arising in caries of the spine. The rapidity of the onset in this boy's case is against such a diagnosis, as are also the lack of any previous history of tenderness over the lumbar spines, rigidity of the spinal column, and the absence of any deep tenderness and swelling in the track of the ilio-psoas muscle on palpation. Confirmation of these points is to be found in the radiogram I have shown you, as there is no trace of caries of the spine to be seen. It is true that on attempting to straighten the thigh he complains of diffuse pain in the region of the iliac fossa, but its position is indefinite at present. The lordosis induced by extension of the hip might be due to an attempt to immobilise this joint, but, as pointed out, he has no sign of disease here: again, it is improbable that it is due to spinal caries, since in this disease the spine is commonly, though not constantly, maintained in a state of rigidity with a view to rest; you may, therefore, assume that this lordosis is present for the purpose of relaxing the ilio-psoas muscle, which is the seat of some lesion in its course. The absence of tenderness over the sacro-iliac synchondrosis, especially when the ossa innominata are pressed towards the sacrum, and of any swelling or œdema here is quite against a diagnosis of disease of this joint, as is also the fact that the pain is in the iliac fossa and not in the track of the sciatic nerve. Acetabular disease, being invariably an extension of hip-joint tuberculosis, must also, in view of the points already mentioned, be negatived.

The acute onset and the pyrexia point to a septic origin, and although there are no other constitutional signs present, you must at least consider the question of perinephritis and appendicitis, and these can easily be excluded. The absence of loin tenderness and swelling is against the former, and the pain of which the boy complains on extension of the thigh is not that which is characteristic of an adherent appendix. It is worth your while to remember the usual situation of the pain induced by this extension in the conditions we have considered. Roughly speaking, you will find that in caries of the spine the pain is referred to the corresponding side of the vertebral bodies slightly below the umbilicus, or to the lumbar region and loins, and is dull and aching in character. In perinephritis the pain is below the costal margin, about half-way between the middle line and linea semilunaris, is dull in character, and tends to run to the loin if extension is persisted in. In the case of an adherent appendix extension of the hip will result in pain referred to the usual site, viz. McBurney's point. Further evidence against appendicitis is to be found in the absence of vomiting, constipation and furred tongue, and of any history of a previous attack. Another acute condition which may give rise to flexion of the hip is the presence of a ureteral calculus impacted at the point at which the ureter crosses the psoas muscle. But in this patient there is a complete absence of any evidence of ureteral colic or urinary frequency. The suggestion made by one of you that the condition may be due to the irritation of a growth implicating the upper portion of the lumbar plexus is reasonable so far only in this, that such a condition *may* thus be produced, but a gradual and not an acute onset would be the rule. When growth occurs in such a situation, the symptoms commonly present are paresis, gradual wasting, occasionally neuralgia, and loss of tendon reflex. I have quite recently had such a case sent to me. The patient was that of a lady who had undergone an operation two years previously for the removal of a tumour growing from the sheath of the anterior crural nerve in the right iliac fossa. Her symptoms when I saw her consisted of weakness of the flexors of the hips, and to a less extent of the extensors of the thigh, and complete loss of knee-jerk on that side. There was, however, little wasting of the thigh muscles, and complete absence of pain and tenderness, and there was no tendency to flexion of the hip-joint. The electrical reactions also indicated the presence of a lesion implicating the upper portion of the lumbar plexus.

With regard to the implication of these nerves it may be stated that inflammatory irritation is more prone to cause muscular contraction, while growth more frequently results in gradual paresis.

Thus far we have considered hysteria, sacro-iliac disease, caries of the spine with resulting psoas abscess, perinephric and appendicular inflammation, impaction of a ureteral calculus, and growths of the lumbar plexus. With none of these condi-

tions do this patient's symptoms entirely agree; but as we have fixed the site of his trouble in the ilio-psoas muscle, we must admit the possibility of a localised septic focus here.

Now, gentlemen, I am aware that such a condition is unusual, but from experience I am also convinced that it is of commoner occurrence than is generally believed. We do not question the possibility of such a thing as a suppurating hæmatoma in the rectus abdominis muscle, in the pectoralis major, or in the extensors of the thigh. Why? Firstly, because we can easily operate upon such superficial parts and prove the existence of the condition; secondly, because we know that here a septic focus is commoner than a tuberculous one; thirdly, because we know that rupture of these muscular fibres is not uncommon. But in the case of the psoas muscle, the site is deep and beyond our immediate observation, the association with spinal caries is very common, and we know little of rupture of this muscle clinically.

Now note the following points: the psoas muscle of all others in the body exhibits just those characters which should render it liable to rupture. It is long; as compared with every other muscle in the body, its fibres are exceptionally delicate, since it contains almost no fibrous material within its sheath; in company with its fellow, the iliacus, it has to sustain almost the entire burden of flexing the thigh on the abdomen, and this at a considerable mechanical disadvantage, since the proximity of its insertion (the lesser trochanter) to the fulcrum (the acetabulum)—it is a lever of the third order—requires that it shall put forth very great exertion to raise a weight so far from this point. It has, moreover, at times to act with great rapidity, as in gymnastics, especially on the horizontal bar, high leaping, hurdle-racing, etc. As regards direct injury, it is in a favourable position to receive bruises, since it lies, backed by bone, along the prominence of the vertebral bodies and transverse processes in its upper part, and along the brim of the pelvis in its middle portion. Lastly, like all delicate muscles, it is richly supplied by blood-vessels which are easily ruptured. The whole of the characters, topographical and histological, and anatomical, are thus favourable to the formation of a hæmatoma either from sudden muscular action or direct injury.

The conditions in the iliacus are essentially different: its fibres are shorter and coarser; it lies in a protected hollow; it is spread out over a flat area and it is far less vascular. It is for this reason that it is in its fellow, the psoas muscle, that the effects of violent action or direct injury are more likely to be met with.

In view, then, of the fact that the patient I have shown you is undergoing gymnastic instruction, that his condition is acute, that it is apparently confined to the ilio-psoas muscle, and is accompanied by pain, tenderness, pyrexia, and flexion of the thigh, and that he has no evidence either clinically or by radiography of localised caries of the spine, I would suggest to you that he is suffering from a localised suppurating hæmatoma of this muscle. But at the same time I would caution you that such a diagnosis can only be a suggestion, since the occurrence of an atypical tuberculous psoas abscess cannot be denied.

As regards the treatment of this boy, you must first of all ask yourselves these questions: Is he in need of operation? If so, by what route must the abscess be approached? What form is his after-treatment to take, and how far is the condition he is suffering from likely to incapacitate him for naval service?

In the first place it is clear that his abscess, whether it be septic or due to a double infection, if not relieved will ultimately spread through the muscle and destroy it entirely, as well as threaten his life, so there is no question as to the need for operation, and although the boy is not urgently ill, for immediate operation, since owing to the delicacy of the fibres of this muscle, and consequently to the slight opposition to the advance of the destruction, he is not likely to appear any worse than he does at present until the entire muscle has been destroyed and the pus is subjected to tension within the sheath.

The method of reaching the trouble is of importance, and such advice as I am able to offer you applies equally well to tuberculous abscesses of this muscle as to the rarer septic ones. In this particular case, since no swelling of any kind is to be felt in the course of the muscle, it is clear, firstly, that the abscess is a small one, secondly, that it is in all probability high up where the muscle belly is thicker and palpation is more difficult, and thirdly, that it is either in the central portion or in the posterior aspect of the muscle. For these reasons

the best, probably the only route by which it should be approached is by a posterior incision along the outer margin of the erector spinæ. The other routes by which a psoas abscess may be reached are quite unsuited to modern surgical requirements. The old method of opening the abscess in the groin is a bad one for these three reasons: it drains, or attempts to drain, the abscess from the highest in place of the lowest level: it is in a septic neighbourhood, and one in which the skin is incapable of standing the maceration which must inevitably ensue, for it is questionable if it is ever possible to obtain primary union here after evacuation of the pus, even when this is purely tuberculous; it is situated at the furthest point from the usual seat of the disease, and, therefore, must be utilised at a time when so much destruction has taken place that a satisfactory result is hopeless.

The second incision which has been employed is that which runs parallel to and above the anterior third of the crest of the ilium and Poupart's ligament. This is thoroughly bad, because not only does it again mean drainage from a higher level than the floor of the abscess cavity, but also it inflicts serious damage to the abdominal muscles and consequently predisposes to an extensive ventral hernia. Moreover, it necessitates stripping up of the iliac peritoneum widely, leaving a huge wound, and infecting a large area of healthy tissue when the pus is tuberculous.

The advantages of the incision I have recommended to you are quite obvious. It deals with the abscess in its earliest stage and at its point of origin which, in the recumbent posture, gives good drainage of the floor of the cavity; it may (although this is not always easy) give access to carious bone when the disease is tuberculous; the skin here is thick and stands maceration well; it does not predispose to hernia and it is far removed from any septic part. On the other hand, the tissues of the back are thick, and the presence of the lumbar aponeurosis makes the edges of the wound inelastic and difficult to retract for exploratory purposes, and in the event of hæmorrhage from the lumbar vessels, it is difficult to catch and tie these.

This abscess, then, must be sought through the back, and if evacuated thoroughly must be closed after thoroughly swabbing out the cavity with tincture of iodine, iodised phenol, carbolic acid, or some such antiseptic. If the disease is tuberculous and early it may in a few cases remain healed. On the other hand, many of these wounds will again require opening up, or will reopen of themselves, in which case drainage becomes imperative, and the cavity must be washed out daily with equal parts of tincture of iodine and rectified spirit, which treatment may be improved upon by first destroying the discharge by the use of a ten-volume solution of (H_2O_3) peroxide of hydrogen. At the time of the primary operation you should never omit the important step of making cultivations from the pus, firstly for diagnostic purposes, and secondly for the provision of an autogenous vaccine in the case of delayed healing. Where such is possible it is of the greatest importance to resort to the inoculation of a guinea-pig, than which there are few more reliable tests for the presence of tubercle.

The question as to whether or no the spine should be immobilised by a plaster jacket depends on the nature of the disease and the character of the patient; in caries of the spine or in the case of a young and restless patient it should certainly be the rule.

Similarly the question of the fitness of the patient subsequently for the naval service for which he is at present destined must rest on the post-operative diagnosis of his condition. If it is proved to be definitely non-tuberculous there will be no question of his capacity for employment; if, on the other hand, it is tuberculous, the question will depend on the complete cure of his local condition, the absence of any resultant kyphosis, and the entire absence of tubercle in his lungs or elsewhere.

Subsequent Note on this Case.

The signs above noted becoming rather more marked the patient was submitted to operation. An incision five inches in length was made along the outer border of the erector spinæ and having its centre opposite the second lumbar spine. The lumbar aponeurosis was divided and a steel director was thrust forwards and slightly inwards to a depth of 2 to 5 inches; this resulted in nothing, and again the attempt was made in an upward direction, but without result; at a third attempt in a downward and forward direction a small abscess was tapped which contained about four drachms of thick yellow pus. A cultivation was made from this, and the

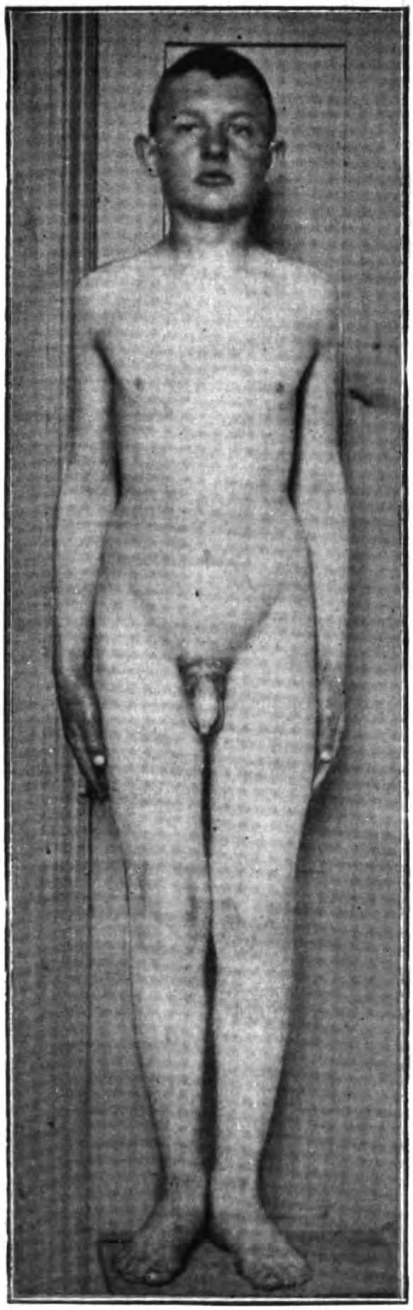

FIG. 3.—Attitude on day of discharge from hospital.

wound and cavity were swabbed out with pure carbolic acid and closed. The wound remained closed for five days when some more pus began to accumulate, and it had to be reopened and a rubber drainage tube was placed in it. From this time the temperature remained normal, the abscess discharging only a small quantity of pus each day. Although my opinion had been against spinal caries throughout, I thought it wise to place the patient in a spinal jacket as he was excessively restless and impatient of restraint. A window was cut in the jacket for the purpose of dressing the wound. It progressed very slowly indeed, but never showed any of the usual characters of a tuberculous wound. It was constantly dressed with peroxide of hydrogen, iodine solution, red lotion, etc., and ultimately it healed up entirely, and for some months now the patient has been convalescent, and has long since discarded his jacket.

Repeated cultivations from the pus proved negative, neither tubercle bacilli nor any other organism being found. The flexion of the thigh and the pain gradually subsided and he is now able to walk perfectly.

December 8th, 1913.

MAP-SCHEME OF THE SENSORY DISTRIBUTION OF THE FIFTH NERVE (TRIGEMINUS) WITH ITS GANGLIA AND CONNECTIONS. By L. HEMINGTON PEGLER, M.D., M.R.C.S., Senior Surgeon, Metropolitan Ear, Nose, and Throat Hospital, etc. Printed in 15 colours, and mounted on rollers for hanging up (size 4 × by 5 ft.), or folded in cloth cover (size 8½ × 11 in.). London: Baillière, Tindall & Cox. Price 21*s.* net.

The intricate distribution of the fifth nerve is a difficulty to remember even by the student fresh from his anatomical studies. How the laboriously acquired facts of anatomy fade from the memory of the practitioner is only too well known. Yet in diagnosing the cause of referred pain a knowledge of the origin and distribution of the numerous branches of the fifth nerve is essential. This graphic presentation of the complex trigeminal system should prove most useful to the student in assisting his reading and dissections. To the practitioner it furnishes a much readier means than the text-book of refreshing his memory, and tracing the cause of pain referred to one part of the head back to its origin in some other part. Analytical tables of the anatomy and physiology of the fifth nerve and its ganglia and connections are also given, and form a compendium of the map-scheme.

A CLINICAL LECTURE

ON

GONORRHŒA AND ITS COMPLICATIONS IN PREGNANT WOMEN.*

By DAVID WATSON, M.B., C.M.,
Surgeon to the Glasgow Lock Hospital, etc.

GENTLEMEN,—At our last meeting we considered the question of the bacteriological diagnosis of gonococcal infection in women, and I might shortly recapitulate the method which we proved to give the most certain results. Swabs are taken from the urethra and from the cervix, the favourite sites of gonococcal activity in the female, smeared on to glass slides, stained according to Gram's directions, and examined with an oil-immersion lens. The urethra, although usually infected, may have escaped, and in taking a smear we must be careful not to carry gonococci into the canal by our manipulations. The meatus and surrounding parts are, therefore, thoroughly cleansed with biniodide solution, and then dried before we interfere with the interior of the urethra. External asepsis being secured, the labia are held apart with the gloved fingers of the left hand. A sterile wool-wrapped probe is passed an inch or so into the urethra, where it is gently rotated, and from this probe a smear is prepared. When there is much vaginal discharge it is almost certain that the cervix is involved, but in no case should the cervix be approached before the external parts and then the vagina have been thoroughly swabbed with an antiseptic solution. After careful disinfection of the whole region, the cervix is displayed through a speculum and itself cleaned. Several wool-bearing probes are used to remove the secretion from the patent cervix without penetrating too deeply into the canal, and when this has been effectively done, a final probe is rotated in the cervix with sufficient firmness to express some contents from the cervical glands. Platinum spoons may be substituted for the probes. By this procedure smears can in most cases be obtained sufficiently free from contaminating organisms to enable the gonococcus, when present, to be demonstrated.

Among the cases being shown to-day are some women several months advanced in pregnancy. Gonococcal infection, while it decreases the probability of conception, does not negative the possibility. Whether a woman is rendered sterile or not depends on the areas involved and the amount of damage inflicted. Gonococcal infection may be coincident with conception, it may follow it, or precede it. Therefore all stages of gonorrhœa—acute, subacute, or chronic—are met with in pregnant women, but as the pregnant state seems to furnish the gonococcus with the conditions which favour its growth, the symptoms are more marked, exacerbations more frequent, and complications more liable to supervene. It is calculated that from 5 per cent. to 10 per cent. of pregnant women harbour the gonococcus, and that from 15 per cent. to 30 per cent. of cases of puerperal fever are directly or indirectly due to the organism.

I need hardly say that pregnancy increases the urgency of the case. Looking at this cervix bathed in offensive pus we see how auto-infection, not only from the gonococcus, but from the *Bacillus coli*, the staphylococcus and the streptococcus, which are all present in this secretion, may arise. These organisms, which at the moment are probably living as saprophytes in the exudation, can readily play a different *rôle* if they gain access to the placental site of a puerperal uterus. Exterminate the gonococcus with its irritating toxins, the inflammatory secretion is no longer produced, and in the absence of the medium on which the saprophytes thrive they can soon be dislodged from the vagina.

Well, how can the gonococcus be destroyed, lurking as it does in the depths of the turgid and œdematous mucosa as well as in its glands and crevices? In the first place there are some general hygienic and therapeutic measures which may be of assistance. Of primary importance in this respect is cleanliness of the external genitals. This is ensured by frequent sitz baths, to which some non-poisonous and unirritating antiseptic is added. We use pynolene, izal, septiforma, and various other cleansing preparations. The patient is warned of the danger of infecting her own eyes with unclean hands, and of the risk of infecting others through contact in any way with her dis-

* Delivered to the Post-Graduate Class at the Glasgow Lock Hospital, September, 1913.

charge. Mothers especially must avoid infecting their own young daughters. Cleanliness prevents the formation of gonorrhœal warts, which sometimes grow to enormous proportions in pregnant women. Balsamics and urinary antiseptics, unless there are symptoms of bladder or urethral irritation, are not of much service, and as they are liable to upset the digestive organs they are seldom employed. Rest in bed is advisable until the condition is well under control, when a limited amount of exercise is not only allowable but beneficial. Constipation must be overcome by diet and gentle aperients such as cascara. No special restrictions of the diet are necessary except as regards spiced foods and alcohol.

Now let us discuss the all-important local treatment. We will first examine two young pregnant women, one just admitted suffering from acute gonorrhœa, the other admitted some weeks ago in a similar condition.

Case 1, the recent admission, shows distinctly how an acute gonorrhœal inflammation is enabled to run riot in the susceptible pregnant state. The vulva is swollen, œdematous and excoriated.

This small aperture into which I pass a probe is the opening of a recently ruptured pseudo-abscess of the left Bartholein gland from which wells gonococcus-laden pus. Clearing away the copious creamy pus, you now notice several red areas of infiltration, particularly around the urethral meatus, and one or two infected crypts and a paraurethral passage which the probe shows to be of some depth. Inserting posteriorly a Sim's speculum and anteriorly this retractor we are able to view the walls of the vagina bathed in purulent secretion and in the posterior *cul-de-sac* a miniature lake of pus obscuring the cervix. Having swabbed the pus gently away, taking care to avoid injury to the tender tissues, we display the large angry-red cervix, and in the patulous os you see the fountain-head of the irritating secretion which induces the sero-purulent vaginal discharge.

Case 2: Examining patient No. 2, who in a like plight was admitted just over four weeks ago, we find that the whole region is practically normal. Only a little thin mucus is exuding from the cervix, and this as well as the urethral smear is now free from gonococci, and has been so for some days.

Our treatment for acute gonorrhœa in pregnancy can, therefore, be best described by detailing exactly how this result has been attained, and how patient No. 1 is about to be treated.

After a prolonged sitz bath, containing about an ounce of kerol, the vagina is carefully and gently swabbed with 1 : 2000 biniodide of mercury solution or 1 : 100 lactic acid. Various other antiseptics may be used, *e.g.* perchloride or oxycyanide of mercury, iodine, formalin, etc. Care must be taken to remove all trace of the antiseptic, and the next procedure is, therefore, to swab well with plain sterile water, and then dry with wool. Collosal argentum or protargol 1 per cent. is then thoroughly applied to each infected area, the urethra, cervix, and every reddened patch or pocket receiving careful treatment with wool-wrapped probes dipped in the solution, or by means of injections through blunted hypodermic needles where probes cannot enter. The parts are again dried, the cervix lightly packed with this powder, one of the pessaries inserted high up in the vagina, and the patient returned to bed. The whole process must be carried out with the utmost gentleness, and can, therefore, only be entrusted to specially trained nurses, otherwise over-manipulation might induce premature labour. We have been singularly free from any mishap of this kind, which can only be attributed to the skill of the sister in charge.

This routine is carried out night and morning for three or four days, when the condition will be found to have greatly improved as regards the quantity of discharge and the presence of saprophytic organisms. We then rely on lactic acid 1 : 100 to 1 : 200 as the only antiseptic for swabbing, and the powder and pessaries as before. The silver preparation is continued for the urethra and all vulvar recesses harbouring gonococci, but the cervix and vagina as a rule only receive lactic acid swabbing and the powder and pessary, whose composition I must now explain. I started in 1909 to use lactic acid bacilli for the treatment of gonorrhœa in women. Several methods of applying this agent have since been tried, and the outcome of our experiments is this preparation. It is a mixture of a liquid culture of lactic acid bacilli and sugar of milk, allowed to dry under aseptic conditions. While the mixture is damp and mouldable, these short rods are punched out of the doughy mass, and after drying are used as pessaries. The remnants are allowed to dry, and

are then crushed into powder form. The preparation has to be made weekly, as the bacilli lose their activity if kept long in the dry state.

There are doubtless plenty of innocuous lactic acid producing organisms which could equally well serve our purpose, and whose vitality would not suffer from being made into tablet form, but we get such good results from our present method that I am loath to change since we have here the facilities for making the product.

But for use in general practice it is highly desirable that a preparation which will keep active for a reasonable period should be put on the market, and I hope that this will soon be accomplished. Meantime the admixture of one of the fluid cultures, *e.g.* lactigen (which is the purest I have examined), with sugar of milk under strict aseptic precautions, will furnish a paste which will serve the purpose excellently.

The rationale of this treatment I can only tentatively explain, but the clinical effects are, as you have seen, highly satisfactory, and I know of no other method of arriving at anything like the same results. To attempt to eradicate the gonococcus during the term of pregnancy by applications of gonococcal antiseptics, however energetic, is well-nigh hopeless. Our use of a preliminary antiseptic treatment shows what benefit can be got in this way, but when a certain stage has been reached no further progress seems to be attainable, and only by continuing indefinitely the antiseptic measures can this improvement be maintained. The point where one can say that absolute cure has been effected, the gonococcus being permanently absent from the smears, is very difficult to realise in women at all, and the difficulties are increased in pregnant women. The addition, however, of the lactic acid bacillus to our therapy alters the whole picture.

Now, by what hypothesis can our results be explained? We use two agents not generally used by others. One, sugar of milk, is known to have an inhibiting action on microbic growth, and doubtless some proportion of the credit must be awarded to the saccharine material. But sugar alone, while helpful, fails in practice to give the full benefit derived from the combination. The other, the lactic acid bacillus, is the more active partner, but it requires the sugar as a medium for growth and for the production of one at least of the agencies by which it effects its beneficial powers, viz. the lactic acid. The gonococci embedded in the epithelial and sub-epithelial tissues are beyond the reach of its action, but the growth of superficial colonies from which the deeply lying organisms are recruited is inhibited and further deep infection is prevented. The continuous antiseptic action of the nascent lactic acid destroys the surface organisms in glands and crevices more effectively than any temporary application of an ordinary antiseptic can be expected to accomplish. But we may accept that there is in addition a healing effect on the protective epithelium, in part directly due to the astringent action of the acid, and in greater part due to the absence of the destructive toxins of the pyæmic organisms.

There is certainly a great reduction in the quantity of secretion poured out by the mucous surfaces, and it may reasonably be inferred that the toxins of the deeply situated gonococci which usually escape in this secretion are diverted into the normal channels of absorption and thus reach the general circulation, where they may be counted on to stimulate the formation of antibodies in quantity sufficient to cope with the gonococci embedded in the tissues, and, therefore, within reach of their action. In this way a natural vaccine therapy is excited and the gonococcus ultimately exterminated.

Whether you will be convinced by this explanation or not, gentlemen, the results you have seen will, I am sure, make you all anxious to test the method whenever you have gonorrhœa to treat in women.

We have under treatment at the present moment one or two cases which illustrate some of the complications to which pregnant women are liable. All the various conditions by which entrance of the gonococcus into the blood-stream can evidence itself may occur during the period of gestation, only pregnancy tends to increase their incidence and their acuteness. So long as a local focus of gonococcal infection exists there is the risk of rheumatic affections of the joints and tendon sheaths, endocarditis and pericarditis, phlebitis, pleurisy, iritis, conjunctivitis, or even grave septicæmia.

The most important element in the cure of these conditions is cutting off the supply of the invading organisms by destroying the foci of infec-

tion in the genital tract. Vaccine therapy is also of value, but there are several points regarding the action of gonococcus vaccine which require elucidation before it can take an assured place in treatment.

In the time at our disposal it is possible to include in our review only a few of the more common of the complications which may be met with in practice.

We have here an example of the rapidity with which gonorrhœal warts (condylomata acuminata) can grow. These masses have appeared within the last three weeks. Pregnancy favours the formation of these growths. Sometimes they develop into large cauliflower excrescences. They usually consist, as in the case before us, of three or four main clumps with enormous numbers of small warts scattered over the vulvar skin and mucous membranes. They may be found on the vaginal wall and cervix and are frequently found around the anus, suggesting rectal gonorrhœa.

The treatment which naturally suggests itself for these large outgrowths is excision, with a free use of the cautery for the small scattered warts; but apart from the risk of operative interference, with its attendant shock inducing premature labour, the result from such treatment is not satisfactory. It is impossible to destroy more than a fraction of the minute warts, and the more thorough the surgical interference the more danger of scar-formation, which will interfere with labour, and may, as I have seen happen, so distort the urethra that incontinence of urine follows. Fortunately we have found that surrounding each separate mass with a wet dressing of lactic acid 1 : 100 causes them to shrivel and drop off usually within a few weeks, leaving no scar whatever. In some cases, especially when associated with syphilis, a fleshy remnant is left which can be snipped off or excised. The small skin warts are touched with undiluted lactic acid and the mucous warts are dusted with bacilli powder until they disappear.

Bartholein abscess, or rather, pseudo-abscess, it being really a suppurating retention cyst, is of frequent occurrence. A small incision to allow of drainage is sufficient, and the cavity is regularly injected with silver solution and packed with gauze until germ-free.

Para-urethral passages are frequently present in women. They are usually blind *culs-de-sac*, but in some cases they communicate with the urethra. They must be carefully swabbed or injected with silver solution, otherwise they will prove a nidus of chronic infection.

When the urethra, in spite of treatment, still gives a positive smear, it is necessary to use the urethroscope to locate and treat any duct or crevice which may not be receiving sufficient of the ordinary application. Skene's ducts, which open into the urethra, may be the source of a continued infection.

Gonococcal cystitis is suggested by the presence of a quantity of pus in the urine as seen in this specimen. A small amount of pus may originate in the urethra, or vulvar or vaginal pus may accidentally be mixed with the urine. To ascertain the condition of the bladder accurately it is necessary for the nurse to cleanse the whole vagina and request the patient to pass urine in two portions. The first specimen contains, in addition to the contents of the bladder, all pus washed out from the urethra, while the second shows the condition of the bladder.

It is not wise to cystoscope a bladder during pregnancy, but all that would be seen in gonococcal cystitis is a varying number of small reddened areas with injected margins scattered over the trigone. The treatment consists in washing out the bladder with a weak solution of permanganate of potash 1 : 8000 to 1 : 4000, or one of the organic silver preparations, *e.g.* albargin 1 : 5000 to 1 : 2000. A urinary antiseptic is also advisable, and in prescribing such it is necessary to know the reaction of the urine. If it is frankly acid or can be rendered acid by administering the acid phosphate of soda in 20-gr. doses three times daily, urotropine is indicated, otherwise boric acid is to be preferred. Sandal-wood oil is also of service both as an antiseptic and sedative, and uva ursi, which is antiseptic and diuretic, is a suitable vehicle as in the following prescription:

℞ Urotropine or boric acid . . gr. x.
Inf. uvæ ursi ad . . . . ℥j

M. Sig.: To be taken three or four times daily. The sandal-wood oil is given in 10-minim capsules three or four times a day after meals.

Extension to the kidneys is a grave, but fortunately uncommon complication. The treatment is ureteral silver injections.

Gonococcal infection of the placenta has been demonstrated as a cause of miscarriage, and penetration of the gonococcus into the amniotic fluid is a rare cause of prenatal destruction of the infant's eyes.

Finally, how can we convince ourselves that a patient is cured and free from risk of recurrence? In the first place there is the absence of all abnormal appearances of the genital tract: no purulent discharge or areas of redness are seen on separating the labia or on displaying the cervix; also no gonococci can be found on prolonged and repeated search either in urethral or cervical smears.

In case of doubt it is possible to excite any latent gonococci into renewed activity, so that they can be found in smears, by slightly irritating applications, such as 1 : 1000 nitrate of silver solution, or by a hypodermic injection of a small dose (five to ten million) of vaccine. If they are present they should be found and destroyed. It is not wise in this case to "let sleeping dogs lie." The intradermic vaccine reaction and the complement-fixation test, if distinctly negative, are of diagnostic value, but it is not yet definitely ascertained how long these phenomena may survive in the absence of the gonococcus. These reactions merely indicate the presence of antibodies in the blood, and it is probable that these antibodies continue to exist for short periods, varying in different individuals, after the final extinction of the organisms. The skin test is easily applied as we perform it here. The needle of a hypodermic syringe containing gonococcus vaccine is insinuated about a quarter of an inch between the layers of the skin, taking care not to penetrate into the subcutaneous tissue, and a few drops of the vaccine are injected. If the needle is properly placed an urticaria-like bleb is seen to form as the fluid enters. Where the reaction is positive an area of intense redness is produced within a few hours, which reaches its maximum in two or three days, remains prominent for several days longer, and thereafter fades. In a negative case a slight redness may result, but this rapidly fades, and has entirely disappeared in two or three days.

The complement-deviation test is, of course, a laboratory procedure. We have been using it experimentally during the past year, and it promises to be of value when we have gained sufficient experience of the technique and of its limitations.

December 8th, 1913.

POST-OPERATIVE EMBOLISM OF THE FEMORAL ARTERY; TWO CASES, ONE BILATERAL, THE OTHER UNILATERAL; ARTERIOTOMY ATTEMPTED IN THE LATTER CASE.

By C. GORDON WATSON, F.R.C.S.

CASE 1.—A woman, æt. 62 years, was admitted to the Metropolitan Hospital on February 27th, 1910, with chronic intestinal obstruction. I opened the abdomen and found an inoperable carcinoma at the hepatic flexure of the colon and performed an ileo-sigmoidostomy. Eighteen hours after operation the patient suddenly complained of numbness and tingling, first in the right foot and then in the left, and of acute pain in both legs. These symptoms were attended by a rapidly established ischæmia of the legs and by the disappearance of the pulse from both femoral arteries. Six hours after the onset of symptoms the legs from the knees downwards were mottled and insensitive, the area of insensitiveness gradually extending upwards towards the groin.

A diagnosis of symmetrical embolism of both femoral arteries was made and the question of arteriotomy discussed, but abandoned on account of the feeble and collapsed condition of the patient. The patient died, apparently as a result of heart-failure, eighteen hours from the onset of symptoms. She had been under observation for some time, but had not manifested any symptoms of vascular disease.

Autopsy.—The right and left femoral arteries were blocked, each by an embolus with some thrombus at the point of bifurcation into superficial and deep femorals. The embolus originated from a small aneurysm in the aortic arch, situated at the site of the obliterated ductus arteriosus, and had evidently been broken into two pieces as it impinged against the sharp edge of the bifurcation of the abdominal aorta. The heart and aorta, together with the iliac and femoral arteries, were removed intact, and the following is the description given of the specimen in the Museum Catalogue at St. Bartholomew's Hospital, where the specimen is preserved (No. 1571 i):

"Two specimens showing a small aneurysm of

the aortic arch, from which a long pendent thrombus has arisen and caused symmetrical embolism of the femoral arteries. The arch of the aorta is in a condition of advanced sclerosis. Attached to its concavity is a part of the left pulmonary artery, showing, in the shape of a small dimple, the obliterated orifice of the ductus arteriosus. The neighbourhood of the aortic orifice of this duct is the seat of a small sacculated aneurysm, within which lay the club-shaped thrombus now seen displaced for purposes of demonstration. The thrombus is $1\frac{3}{4}$ inches long, and has a partially detached fragment hanging from it near its extremity. The extremity itself is jagged, and it is obvious that a part has been recently broken from it. The lower specimen consists of the bifurcation of the aorta and its continuations, and shows the embolisms of the femoral arteries which resulted from the impaction of detached pieces of the aortic clot. The common iliac arteries, together with the terminal portion of the aorta, show extreme sclerosis. From a point some 2 inches above the origin of the profunda femoris, downwards, each femoral artery is occluded by a firm thrombus. It is not possible to say with confidence how much of these thrombi is embolic in orgin, and how much depends upon consecutive thrombosis."

CASE 2.—A man, æt. 60 years, was operated on in private for carcinoma of the larynx by Mr. F. A. Rose. The entire larynx was successfully removed on December 14th, 1911. A fortnight later the patient was suddenly seized with pain and tingling of the left foot. This was followed by an ascending pallor, loss of sensation and power in the left leg. When seen by me six hours after the onset of symptoms no pulse could be felt in the femoral artery below the bifurcation, but iliac pulsation could be felt. The foot and leg up to the knee were cold, pallid and insensitive.

Immediate operation was decided on, and I exposed the common femoral artery. Pulsation ceased at the bifurcation and I decided to open the artery with a view to embolectomy. Whilst raising the artery from the femoral sheath for the purpose of applying a Crile's clamp, pulsation suddenly returned in the superficial femoral artery. I then exposed the superficial femoral at the lower end of Hunter's canal, as I was unable to satisfy myself that pulsation could be felt there. I found that the vessel pulsated as far as the middle of the popliteal space, and closed the wounds.

Next day some warmth had returned to the foot, but no pulsation could be felt in the tibial arteries. Gangrene of the foot eventually became established, and on January 4th, 1912 (a week later) I amputated the leg through the condyles of the femur.

The artery was completely blocked at the bifurcation of the popliteal by thrombus, which extended down into both tibials. I was unable to identify a definite embolus.

The patient made a good recovery from the amputation, and is now, nearly two years after the operation of layngectomy for carcinoma, free from recurrence, and in good health. There is no evidence of vascular disease.

REMARKS.

Despite the remarkable experimental results which Carrel has secured, arteriotomy for embolism has not proved successful in many instances. The two cases described above illustrate, in the one case, the fact that the condition of the patient with post-operative embolism may be quite unsuitable for operation, and in the other, that care is required to prevent dislodgement of an embolus during the preliminary stages of the operation of arteriotomy. Had I first taken steps to compress the superficial femoral below the site of the embolus, before compressing the artery above, I should have been able at any rate to give embolectomy a trial.

To expose the popliteal artery at its bifurcation and to remove an embolism would be a difficult operation. It is a matter of regret that having accidentally displaced the embolus from the bifurcation of the common femoral to the bifurcation of the popliteal, that I did not immediately attempt, by massage along the course of the popliteal artery (without exposing the vessel), to break up the clot into small particles (*pétrissage*). This simple proceeding should stand a good chance of success when carried out immediately after the lodgment of an embolus.

In the first case of bilateral embolism some doubt existed in my mind as to the situation of the embolus. Was the embolus at the bifurcation of the abdominal aorta, or were the femorals simultaneously blocked? The abdomen was well covered and somewhat distended, and I was un-

able to palpate the iliac arteries. Despite the desperate condition of the patient, I think I should have exposed one femoral under local anæsthesia and established a diagnosis, and then, if a diagnosis of femoral embolism was found to be correct, I should have attempted to remove the embolus, first on one side, and then on the other.

It may well be asked which method offers a better chance of success as regards gangrene—exposure of the artery and an attempt to break up the clot by massage, or incision into the artery and an attempt to re-establish the channel by removal of clot.

There are two important factors to consider, viz. (1) the condition of the lining of the vessels; (2) the length of time that the vessel has been occluded.

(1) If the patient is old and suffering from arteriosclerosis, subsequent thrombosis is likely to occur, even if an embolus is successfully removed, and consequently *pétrissage* should offer more chance of success, even though resulting in the blocking of vessels lower down.

In these cases immediate massage of the affected part should first be tried, and if unsuccessful the artery should be exposed and an attempt made to dislodge the embolus by *pétrissage.*

In the case of a patient under middle age, with healthy vessels, arteriotomy conducted on careful lines should, I think, be the method of choice.

A proximal clot rapidly forms as far up as the nearest branch. This fact must not be forgotten in operative procedures.

(2) The lodgment of an embolus in an artery is rapidly followed by thrombosis, the extent of which will depend on the condition of the heart and arteries and on the size of the vessel. Consequently, the earlier the attempt at embolectomy, the greater the likelihood of success. At the same time massage is less likely to succeed once thrombosis has become established.

On reference to the literature of the subject I find that a successful case of arteriotomy of the femoral artery was reported at the Académie de Médicine.

A man, æt. 38 years, was admitted into the Saint Antoine Hospital suffering from cardiac dyspnœa with mitral stenosis. One morning when getting out of bed he felt a severe pain in the left calf, which he compared to intense cramp. The limb became powerless, and when seen three hours later the foot and leg were cyanosed, livid and "marbled" in places by white ischæmic patches. Sensibility was abolished up to the knee and movements of the foot and leg were impossible. No pulsation could be felt in the limb except in the femoral artery in the upper part of Scarpa's triangle. Six hours after the onset of symptoms the femoral was exposed by M. Labey. Under local anæsthesia a bifurcated clot was removed, which extended from the common into the superficial and deep femorals. The artery was sutured and the blood-current re-established. Recovery was uninterrupted.

Key* was successful in removing an embolus from the common femoral seven hours after the onset of symptoms. A man, æt. 42 years, with mitral stenosis, was suddenly seized with acute abdominal pain and blood-stained diarrhœa, followed by vomiting, sweating, and collapse. Perforation of a gastric ulcer was at first suspected, but the subsequent history pointed to mesenteric embolism. This was followed three weeks later by sudden acute pain in the left popliteal space followed by the classic signs and symptoms of femoral embolism. A large bifurcating embolism was removed from the common femoral, and perfect recovery followed.

Murphy, of Chicago,† in 1909, successfully dislodged an embolus from the common iliac artery of a woman, æt. 41 years, and restored the circulation of the limb. He incised the common femoral and restored the lumen of the common iliac by passing a catheter into the aorta. The patient came under treatment four days after an embolus had lodged at the bifurcation of the aorta. Dry gangrene had commenced with a line of demarcation four inches below Poupart's ligament, but circulation in the femorals was re-established and enabled amputation to be performed at a lower level than would otherwise have been necessary.

M. Paul Claisse‡ has recorded a successful case of *pétrissage.* A woman, æt. 33 years, with heart

* 'Bulletins de l'Academie de Médicine,' December 19th, 1911, p. 358.

* 'Wien. med. Woch.,' June 3rd, 1913, p. 936.

† 'Journ. of Amer. Med. Assoc.,' May 22nd, 1909, p. 1661.

‡ 'Bulletins de la Soc. Méd. des Hôpitaux,' June 10th, 1910, p. 766.

disease, was suddenly seized with intense pain in the calf of the left leg. Two hours later pulsation was felt in Scarpa's triangle, but not below it. The left foot was very cold. In order to break up the clot energetic massage of the inner side of the thigh was carried out at once. After twenty minutes the signs of embolism had disappeared and the foot had regained its normal temperature.

M. Pierre Merklen* has recorded a very similar case of successful *pétrissage* for femoral embolism. For two hours without interruption energetic massage of the femoral, popliteal and posterior tibial arteries was employed to the limb, which had suddenly become cold and lifeless, until the circulation was restored.

Cases have been recorded in which an embolus has been dislodged spontaneously. In one case† an embolus was first arrested at the bifurcation of the aorta, producing paraplegia. On the next day it had reached the external iliac artery of one side, bringing relief to the opposite limb. On the third day the embolus descended to the popliteal.

December 8th, 1913.

BRONCHIAL ASTHMA: ITS PATHOLOGY AND TREATMENT. By J. B. BERKART, M.D., late Physician to the City of London Hospital for Diseases of the Chest. Revised and Abridged Third Edition. Pp. 150. London: Henry Frowde, Oxford University Press. Price 5*s*. net.

The author has for many years advocated a conception of bronchial asthma different from the one currently accepted. The promiscuous use of the term "asthma" rendered it necessary for him to define at the outset the form of disease which is commonly called "bronchial asthma." Whatever views on its pathology may be held this much will be generally admitted—that it consists of recurrent attacks of dyspnœa, which are due to a transient endogenous obstacle in the bronchial "tree." The question, therefore, is, What is the nature of that transient obstacle? The traditional theory, still commonly held, is that the obstacle proceeds from a spasm of the bronchial muscle. That theory, however, was introduced into modern pathology on no other foundation than the mere presence of the bronchial muscles. Experiments on animals subsequently undertaken have not established it, for no recurrent attacks of dyspnœa have ever been produced. The dyspnœa that has been produced occurred in circumstances which have not the remotest analogy with what takes place in man, and its clinical characteristics are not those observed at the bedside.

To arrive at an adequate conception of the nature of attacks of asthma, as above defined, the author considers the following clinical facts: (1) No one in perfect health becomes suddenly subject to that form of disease. (2) The attacks are observed only in individuals who have deformities of the chest—deformities which existed before the onset of the symptoms. (3) The attacks have always been preceded by acute or protracted inflammatory changes of the lungs, from which recovery has been imperfect. (4) The attacks are always associated with catarrhal changes. (5) The post-mortem examinations always show chronic lesions, the effect of which is to reduce the respiratory surface and to diminish the elastic power of the organ. Thus the conclusion is reached that the dyspnœal paroxysms have an anatomical substratum, which consists of some form of bronchial catarrh. Judging by their peculiar clinical manifestations the catarrhal changes are of a chronic kind, and differ in important respects from other analogous affections.

This conception of the pathology naturally leads to a corresponding therapy. The obvious indications are to promote the expectoration of the accumulated morbid products and to prevent their formation. The most important measure Dr. Berkart considers to be rest, which has a salutary influence on the nutrition of the bronchial mucous membrane. He insists on scrupulous cleanliness of the oro-nasal cavities, which may be promoted by stuffing up the nostrils a normal saline solution with the addition of a little liq. hydrarg. perchlor. He advises warm baths, which produce derivation from the bronchial mucous membrane. Potassium iodide may be useful as an expectorant between the attacks, but it is employed much too indiscriminately. If the bronchi are filled with a loose gelatinous secretion an emetic of sulphate of zinc may be given. If other measures fail or the dyspnœa returns at short intervals the obstruction

* 'Bulletins de la Soc. Méd. des Hôpitaux,' November 6th, 1903, p. 1180.

† 'Semaine Médicale,' 1888, p. 65.

to the bronchi may be speedily relieved by injections of anti-diphtheritic serum (1500 units), which has a solvent action on fibrinous exudations. In the severe paroxysms nothing gives such rapid and speedy relief as the hypodermic injection of morphine. Inhalation of the smoke of powders and cigarettes—so much used—Dr. Berkart condemns as irritating to the bronchial mucous membrane. The effect is temporary, and due to the production of a powerful cough by which a few pellets of mucus are expectorated, but the irritating fumes start fresh inflammation, while the stramonium which they all contain adversely affects the nervous system.

It will be seen that this is an original book, which should prove useful to the practitioner in dealing with so troublesome an affection.

Sclero-corneal Trephining in the Operative Treatment of Glaucoma. By Lieut.-Col. Robert Henry Elliot, I.M.S., Madras, S. India. London: George Pulman & Sons, Ltd., 1913. Price 7*s.* 6*d.* net.

The past few years have seen a renaissance in the operative treatment of glaucoma, for it is safe to say that at no period since the introduction of iridectomy by von Graefe in 1856 has the subject of glaucoma been so much studied from the practical point of view. The treatment of glaucoma by sclerectomy is certainly not new, but the tech nique within the past few years has reached a point approaching perfection. Nowhere are the minute details of the various methods employed by different operators more fully and more carefully described than in the present monograph. Consisting of twelve chapters contained in 111 pages, the methods practised by other operators, as well as his own, are lucidly set forth by one whose experience of the disease and its treatment in South India is unique. We have read the book with great interest, and can recommend it to every operator who has to deal with this dread disease. The book is well printed and illustrated, reflecting credit on author and publishers. It is undoubtedly a valuable contribution to modern ophthalmology.

The Practitioner's Practical Prescriber and Epitome of Symptomatic Treatment. By D. M. Macdonald, M.D., M.O.H., Leven, Fife. Pp. 198. London: Hodder and Stoughton, 1913. Price 5*s.* net.

This is eminently a book for the general practitioner. It contains just the information he requires; it will slip into the pocket and be useful for reference. Formulæ are given for all ordinary maladies. There are chapters on practical points in prescribing, common incompatibles, therapeutic brevities, and diet tables. There is also a chapter on emergencies in everyday practice, from which the youngest practitioner may learn much and the advanced one refresh his memory. An important feature is that the drugs included are in the B.P. The book will thus be especially useful to those who practise under the Insurance Act, in view of the pronouncement of the Commissioners on proprietary remedies. The diseases are given in alphabetical order. The style is commendably terse, so that the information can be obtained in the readiest possible manner. An excellent *vade mecum* for the general practitioner.

Clinical Electro-cardiography. By Thomas Lewis, M.D., D.Sc., F.R.C.P., Assistant Physician and Lecturer on Cardiac Pathology, University College Hospital. Pp. 120. London: Shaw & Sons, Fetter Lane.

This is a statement of our present knowledge in the sphere of the above subject. Dr. Lewis has already written on the 'Mechanics of the Heart Beat' and 'Clinical Diseases of the Heart Beat,' and in this book both these works are supplemented by use of electro-cardiograms. The discovery of the instrument used, the string galvanometer, is due to a Dutch physiologist, Einthoven. It consists of a powerful electro-magnet, the poles of which are closely approximated, and of an extremely delicate conducting fibre of silvered quartz. The minute currents are led through this film, and the magnified shadows are projected by means of an optical system on the horizontal slit of a camera, while the movements are photographed on a sensitive plate which travels behind the slit. The electro-cardiograms are specially interesting in the study of partial and complete heart-block. In analysing disturbance of the myocardium this mechanism opens up new and wide possibilities.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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ON ERRORS OF DIAGNOSIS IN EAR DISEASES.*

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IN all branches of medicine and surgery practitioners are prone to make occasional errors in diagnosis. *Hominis est errare*, and physicians and surgeons are but men. To avoid them we must be ever on the alert, and this is beyond the power of man. Even great Jove nods at times, and the man who boasts that he is never wrong is guilty of gross inexactitude. The best we can do is to leave no stone unturned in making our diagnosis. We must endeavour to follow the windings of the case by careful attention to its history, and to ascertain the patient's real condition by close examination. Then, so long as we can bring a logical reasoning to bear upon our facts, we can do no more, but to do this we must be sure of our facts, and these facts can be ascertained only by omitting no part of our clinical investigation. Whenever I find that I have made an error of diagnosis, I conclude that I have incorrectly observed, and I believe that most errors of diagnosis are traceable to incorrect observation. Observation and deduction are the paths by which we reach our opinion as to a patient's condition, and for that opinion to be the right one our observation must be correct and our deductions must be logical.

Hence the introduction to any discussion upon errors of diagnosis in ear disease must be to enumerate the methods at our disposal for correct observation. These may be divided into the investigation of the patient's history, the physical examination of his ears, nose and throat, and of his body generally, and the functional examination

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of the organ whose disease we are endeavouring to make out. And let me observe here that the secret of a good specialist lies not only in his careful and skilled examination of the organ with which he has most to do and the experience he has had in making deductions from the result of such examination, but also in his ability to look at his patient as a whole and not as an organ only. Let me illustrate my meaning by an example. Two patients come to us complaining of early deafness, and we find that in both cases this deafness is due to catarrhal middle-ear disease, which is in itself traceable to naso-pharyngeal catarrh, dependent upon a deflected septum which causes the nose to be imperfectly drained. We treat both patients on the same principle—that is to say, we correct the nasal septum by the modern operation of submucous resection, and we remove the enlarged posterior ends of the inferior turbinals. Yet we find that whilst one patient recovers speedily and his ears do not trouble him again, the second patient goes from bad to worse, and, despite careful treatment by inflation, intra-tympanic injections, oto-massage, etc., progresses to chronic deafness of marked degree. Why should two such cases, apparently so similar, make such markedly dissimilar progress? In the majority of cases we shall find that the secret lies in want of attention to the patient's general condition. The patient who got so well so speedily and satisfactorily, and who speaks to his friends so highly of our skill, was an uncomplicated case, whilst the second, who lets his friends know that "so-and-so is no good," is one whose mucous membranes will not recover themselves because of some toxæmic condition. He has a dirty tongue, a muddy complexion, brownish discoloration of the skin at the oral angles, and suffers from chronic constipation and consequent alimentary toxæmia from intestinal stasis. Pay attention to his digestive tract, and success will follow.

When inquiring into the history of a patient suffering from disease of the ear, omit nothing that will throw light upon previous illnesses, mode of life, alcohol, tobacco and other drug habits, residence abroad, mode of onset, presence or absence of vertigo, tinnitus, pain, otorrhœa, headache, paracusis Willisii, presence or absence of deafness in other members of the family.

Make your physical examination with care, and do not allow your patient to leave until you are satisfied that you have observed correctly the condition of his ears, nose, naso-pharynx, pharynx, mouth, tonsils, fauces and teeth. When he has a discharge in the meatus, never neglect thoroughly to cleanse the meatus, and to observe well the condition of both meatus and tympanum. Neglect of this obvious precaution may lead to the error of mistaking a severe external otitis for middle-ear disease or to the failure to recognise the presence of a polypus. If any tenderness is present ascertain its site; tenderness over the mastoid should be sought by exerting a gradual firm pressure with the thumb over the site of the antrum and over the tip of the process, whilst watching the patient's eye for an involuntary spasmodic closure. Tenderness in front of the tragus usually means inflammation of the external meatus—under the ear, Eustachian tube involvement.

Carry out your functional tests with equal care, remembering that your diagnosis should rest upon the combined results given by both physical and functional investigations. Follow a routine method, you are then less likely to miss something. Ascertain with the tuning-fork the Weber, Rinne, and Gellé reactions, take the bone-conduction and find out whether there is any reduction in the low- and high-tone limits of hearing, and see at what distance the acoumeter, voice and whisper can be heard. Bear in mind that a slight loss of bone-conduction does not necessarily mean nerve-deafness, and that even a marked loss is not similarly indicative unless it is accompanied by a diminution of hearing for high tones. Also remember that the effect of age may mask a middle-ear deafness by affecting the result of Rinne's test, and that, whilst reduction in the high-tone limit means internal ear involvement, a reduction in the lower-tone limit indicates middle-ear deafness. In the latter case remember that the amount of this reduction in hearing for low tones is a valuable guide to prognosis, the outlook as regards treatment being worse in proportion to the amount of this reduction, and that when the patient cannot hear a C 128 tuning-fork by air-conduction, the chances of marked amelioration begin to be slender. Lastly, bear in mind that there is a condition of physiological deafness, that is to say, every person has not always the same degree of normal hearing.

After these tests of cochlear function come those for that of the vestibular apparatus. Theoretically, every case should be examined for nystagmus, and by the caloric and Romberg's tests. In this country we are not usually so exacting, but on the Continent functional testing is more thorough. Here, however, it is customary only to make a full investigation of the labyrinth when we suspect it from the symptoms to be diseased. On the Continent no otologist would think of performing the mastoid operation unless the case had previously been so investigated, and there can be no doubt that this is the right and scientific course to pursue.

Now let us take the different parts of the ear—external, middle and internal—and see in what conditions errors of diagnosis may occur and how they may be avoided. First, let us take the reflex phenomena that may arise in connection with the ear. The first of these is a reflex otalgia, which may be due to disease of the teeth, tongue, or fauces, the commonest cause being a carious tooth. I have known normal ears to be treated by all sorts of methods for pain when the real source lay in dental caries, and a visit to the dentist would have put everything right. In such cases it must be borne in mind that toothache may be entirely absent, the sole manifestation of pain being in the ear on the side affected. Such reflex pain occurs through the fifth nerve.

Other reflex phenomena that may arise from the ear itself are ear cough, hiccough, syncopal attacks, taste reflexes, etc., and I must refer you to text-books upon the ear and its diseases for further information on the subject, merely reminding you that cases of persistent cough and also of hiccough have been reported in which treatment has been unavailing until the real source of the irritation in the ear was detected.

The other conditions met with in the external ear likely to lead to errors of diagnosis are collections of cerumen, otitis externa, and early malignant disease. One would hardly believe that cerumen might give rise to confusion, but I have actually had cases brought to me in which deafness was due solely to this cause, and in which unskilled syringing had only served to drive the plug more deeply in the meatus, and so to accentuate the deafness, and even give rise to pain which was not there before. Other cases have come in which the deafness was presumed to have been due to wax, and the ear syringed when none was there, naturally without any resulting relief. The moral of such cases is that no case should be treated by syringing without ascertaining first that a plug exists, and, further, if the surgeon is sure that one is present, to exercise care in removing it by skilful washing out, with or without preliminary softening. Could you see some of the letters that I have received with patients, in which most circumstantial, even weird, accounts of the appearances of the tympanic membrane have been given, and in whom the meatus has been proved to contain plugs of wax completely obscuring any view of the deeper parts, you would be surprised that such remarkable inaccuracies could occur in the diagnosis of so simple a condition.

Ceruminal accumulations may present very varied appearances. They may appear as hard, black, glossy plugs, or as white softish masses, with every gradation between these two extremes. They may be intermixed with considerable masses of dead epithelium, and in such cases require careful loosening by means of peroxide of hydrogen before removal. Very fluid wax may be mistaken for a discharge, especially when it becomes foul, as it may do in school-children of the lower classes. Or it may be mixed with a middle-ear discharge in a mushy, foul mass. Both these considerations require a little ordinary care to avoid possible error.

Occasionally one finds a plug of cerumen accompanied with considerable pain, especially when efforts are made to remove it. Sometimes this is due to its hardness and its pressure upon the sensitive tympanic membrane, sometimes to pressure upon granulations springing from the meatus or middle ear. I remember a case in which the plug lay over a bed of granulations round a small sequestrum in the bony meatus. Very occasionally the pain is sufficiently severe to necessitate the removal of the plug under an anæsthetic. These cases also require a little care in diagnosis.

An acute external otitis may be either diffuse or circumscribed, the latter cases being those usually of aural furunculosis. These may be accompanied by so much swelling as to mask the actual condition and simulate a diffuse inflammation. A leech applied just in front of the tragus, and followed by dry heat, will usually reduce the

swelling sufficiently to allow of a diagnosis to be made correctly. Both cases must be distinguished from acute middle-ear inflammation. In acute otitis externa the deafness is usually less marked than when the inflammation involves the middle ear. Moreover, in the latter case there will be usually indications of infection *viâ* the upper air-passages, there will be much less swelling of the meatus, and the tympanic membrane will show signs of acute inflammation with bulging in some part, as a rule in the posterior segment.

During the early part of this year there has been a number of cases of external otitis in which the membrane itself was involved, and showed a tendency to somewhat prolonged desquamation. These cases presented occasional difficulty of diagnosis unless investigated with care.

Malignant disease of the external ear does not usually present any special difficulties in diagnosis when it affects the auricle. Epithelioma is the most common, other forms being rare. Malignant disease may, however, occur primarily in the meatus, either as so-called endothelioma commencing in the sebaceous and ceruminous glands, or as epithelioma, which is the more common. Epithelioma usually commences in the deep parts of the canal owing to the continued irritation of a long-standing discharge from the middle ear. As, however, the growth is usually not recognised as such until it has already progressed to some extent, it is often difficult, or impossible, to say accurately whether it commenced in the meatus itself or in the middle-ear. In cases of very long-standing otorrhœa, especially in oldish people, with rapidly recurring, friable and exuberant granulations and profuse fœtid discharge, malignant disease is to be suspected, and the case should be submitted to microscopic examination. In such cases one should be on guard against errors of diagnosis.

Passing now to the middle ear, we have to consider both suppurative and non-suppurative conditions. A discharge may be due either to external otitis or to middle-ear disease. Error is unlikely so long as due care is exercised in physical examination. In investigating middle-ear discharge attention must be paid to the presence or absence of granulations, mastoid and other complications, and a whole lecture might be written on this part of our subject. It is only by careful examination and cleansing that one can avoid possible error. As has already been said, no case should be permitted to leave the consulting-room until a thorough cleansing has been carried out, so that all parts of the ear can be seen clearly. Large granulations or polypi may be hidden under profuse discharge, and may escape notice if this precaution be not exercised. It is important to ascertain the origin of a granulation, and to determine whether caries is present. It may be said that granulations in the ear may be classified roughly into three divisions—those due to neglect, those due to caries, and those due to malignant disease. The first are usually large, filling the meatus and sometimes protruding from its aperture. Once removed and proper after-treatment instituted, the case will clear up rapidly. Granulations due to caries are usually smaller, recur on removal, and grow from ossicles or the inner tympanic wall, discharge being usually fœtid. Fœtor is, however, never a symptom to be replied upon, as it may be due simply to retention of discharge. Granulations due to malignant disease recur rapidly, are exuberant and very friable, and bleed readily.

A word may be said here as regards the diagnostic guidance obtainable from the position of a perforation of the tympanic membrane. These may be found in the membrana tensa or in Shrapnell's membrane. It is a useful rough indication that, in the latter situation, a perforation which has existed any length of time is due to caries of the body of the incus, possibly with mastoid involvement, when it is in the posterior part of Shrapnell's membrane; to caries of the head of the malleus and of the attic-wall when it is in the centre; and to attic disease associated with infection through the Eustachian tube when situated anteriorly. Perforations in the anterior segment of the membrana tensa, especially when low down, are usually associated with Eustachian involvement. A posterior superior perforation of the membrana tensa usually means caries of the long process of the incus, and is often associated with mastoid trouble.

In acute and subacute inflammation of the middle ear I have known a highly congested and bulging membrane to be mistaken for a granulation, the real condition being revealed upon attempting to remove it. I have also seen a bulging of Shrapnell's membrane to be similarly mistaken.

Mastoid disease is likely when, after careful

cleansing and drying, pus reappears in the upper and posterior part of the tympanum, or can be sucked out by the Siegle's pneumatic speculum.

In cases of discharge of some standing, the appearance of white, pearly looking skins coming from the attic and near the *aditus ad antrum* are indicative of cholesteatoma. They should always be removed as far as possible, and the tympanum inspected thoroughly. This suspicion of cholesteatoma is confirmed if the patient suffers from periodic headache and attacks of pain from hygroscopic swelling of the accumulations of these skins.

In dealing with cases of chronic suppuration a warning must be given as to treatment. We must not be content with merely treating the condition by way of the meatus, important as this is. The tympanum must be attacked from the side of the naso-pharynx as well, or the result will be disappointing. Any abnormal condition in the nose and throat must be treated, adenoids removed, and the naso-pharyngeal ostium of the Eustachian tube kept clear of infection. Regular inflation must be practised in order that discharge may be blown out of the tympanic opening of the tube, and applications made to the latter region through the meatus. For this purpose it will be found that a 50 per cent. solution of chromic acid, or, more occasionally, a 2 to 5 per cent. solution of nitrate of silver, applied on a small cotton swab, is most useful. Another useful point in adults is the regular inflation by the catheter with iodine and camphor vapour, which assists in keeping the naso-pharyngeal part of the Eustachian tube free from infection.

Sometimes, in children, cases are seen in which, after the removal of adenoids, a chronic middle-ear discharge does not yield to treatment, or, if it does, reinfection occurs repeatedly. Other causes being eliminated, it will practically always be found that the naso-pharynx is at fault, reinfection occurring by the Eustachian tube. This is due either to imperfect removal of the adenoids or to want of efficient after-treatment of the nose and naso-pharynx. This is a matter to which I have already alluded in previous lectures at the Polyclinic. In the case of imperfect operation, it is the central mass of adenoid which has been removed, while portions of the hypertrophy have been left in Rosenmüller's fossæ. It is from these extensions into the fossæ that the reinfections take place. In the second condition—want of efficient after-treatment of the nose and naso-pharynx—it must be remembered that efficient removal of adenoids is not enough unless the infective rhinitis which the growth has set up is treated, for the nose then reinfects the naso-pharynx, and, through that region, the tympanum.

Before leaving the subject of middle-ear suppuration, allusion must be made to tuberculosis. Primary tuberculosis of the temporal bone is most frequent in children, and may attack it either as a mastoid osteitis, in which case it is conveyed through the blood-stream, or as an infection of the tympanum through the Eustachian tube, usually by the passage of infected material into the tube by regurgitation of food, especially milk. The Eustachian tube in the child is wider and more patent than in the adult. In early cases of the latter variety the tuberculous foci may sometimes be seen through the semitransparent tympanic membrane on the inner wall of the tympanum as small, greyish spots, or as similar spots in the substance of the membrane itself. Perforation soon occurs, and painlessly. In very early stages the tubercle bacilli may be found in the discharge, but they speedily disappear with the occurrence of other micro-organisms, which mask the original infection. Speaking generally, the characteristic features of middle-ear tuberculosis are: painless onset, multiple perforations seen in a sodden, pale membrane, which speedily coalesce with considerable destruction of tissue, the appearance of flabby, pale, gelatinous-like granulations with a thin, irritating, but sometimes creamy discharge, and early facial paralysis.

When the disease starts as a tuberculous osteitis of the mastoid, the case is similarly characterised by its comparatively painless nature. Swelling and fluctuation appear over the mastoid, and this, when opened, yields a creamy pus, with a cavity lined by characteristic granulations.

Early diagnosis is important in all cases of tuberculosis of the temporal bone, as treatment—operative and general—must be instituted promptly if hearing and life are to be saved. Of course advantage should be taken of the clinical tests for tuberculosis.

We must now proceed to consider some of the problems of non-suppurative middle-ear disease which may lead to errors of diagnosis. The necessity for careful physical and functional

examination of the patient is especially to be insisted upon here. Ordinary catarrhal deafness may be divided into four stages. The stage of congestion and catarrhal inflammation, when the patient notes that he is deaf whenever he has a cold, but the deafness disappears when the cold has gone; the stage of organisation of inflammatory products, when each attack of deafness does not clear up completely, but leaves him a little more hard of hearing; the stage of fibrous tissue formation, in which the deafness has become well established, and which passes by insensible gradations into the fourth and last stage of post-catarrhal cicatricial contraction. Now there are certain points about all these stages which are concerned with diagnosis, and especially with prognosis. The characteristic tuning-fork reactions of middle-ear deafness are: a negative Rinne, increased bone-conduction, and a reduction in the limit of hearing for low tones. It is beyond the province of this lecture to enter into any discussion as to the reasons for these reactions, and they must, therefore, be taken here as facts. Their complete development, moreover, is not attained until the disease has reached a certain stage. Now, in all cases of middle-ear catarrh, with certain exceptions, the earliest stage is generally confined to the Eustachian tube alone, changes in the middle ear itself, save, of course, in the tympanic membrane, being later in appearance, and dependent upon extension of the catarrhal processes up the tube into the tympanic cavity. There are, however, cases of slow, chronic progressive deafness in which, without any special history of recurrent colds in the head, the tympanic cavity becomes invaded gradually, *viâ* the Eustachian tube, by extension from the pharynx, and these are sometimes distinguished by the name of "chronic dry catarrh." Such cases are sometimes difficult to distinguish from the condition known as otosclerosis, to which I shall presently refer.

Let us take first the ordinary catarrhal case, beginning with Eustachian tube deafness. These always begin in the nose and naso-pharynx, sometimes from untreated adenoids, sometimes from abnormal conditions in the nose proper. From my experience I am led to believe that one of the most common causes in the latter category is an abnormal septum nasi. The want of proper nasal drainage from a deflected septum or a posterior septal bony spur sets up a chronic catarrh in one or both of two ways, either by rendering the nasal mucosa less resistant to the micro-organisms of a common cold, whereby repeated attacks of acute catarrh occur, each of which leaves its mark on the mucous membrane, and accentuates the nasal obstruction in the nose and the bad drainage, so resulting in a vicious circle, or by the irritation of a posterior deflection or bony spur causing enlargement of the posterior ends of the inferior turbinates. In either way, the naso-pharynx partakes in the catarrhal condition, partly by extension, partly by the effect of irritating secretions poured into it through the posterior nares. In the case of enlarged posterior ends the effect is particularly striking, because being quite close to the naso-pharyngeal ostia of the Eustachian tubes, and because the glandular elements of the turbinate share in the hypertrophy and so pour out an increased secretion, the Eustachian cushions and orifices are continually bathed in this secretion and kept in a condition of constant irritation. This can be seen by means of the Holmes's electric naso-pharyngoscope. The importance of all this is very great, for it means that in all cases of commencing middle-ear catarrh (and it must be remembered that the Eustachian tube is only part of the middle-ear tract, and that, as an American otologist has put it, the real orifice of the Eustachian tube is at the external naris), if there is any abnormality in the nose and naso-pharynx, every effort must be made thoroughly to remove it, if the patient is to be saved from progressive deafness. It is not enough, in cases of deflected septum and posterior end hypertrophy, to try to palliate matters by nasal sprays and washes; the cause has to be remedied by operation; the former is mere *dilettanti* temporising, the latter is efficient treatment, a very great difference. I am inclined to think that, even now, many practitioners do not realise to the fullest extent the importance of thus acting. If prompt and efficient measures are taken at the outset the progress of the catarrh can be arrested. I have clinical experience that assures me it is so. Treatment of the ultimate results of catarrhal middle-ear deafness are at present so unsatisfactory that it is our duty, as indeed it is in every other department of medicine, to look rather to prevention as our great aim.

At first the results are, as I have said, wholly

those of Eustachian obstruction, the diagnosis of which lies in the early history of deafness with colds, an indrawn membrane, probably otherwise normal at this stage, and evidence on inflation of a lessened patency of the tube, inflation bringing about considerable improvement. Tuning-fork reactions show that the deafness is not yet marked. If one ear only is affected, as is often the case, Weber's test will be positive, the sound being referred to the affected ear. Rinne's reaction will be negative to a varying degree, and probably only for a low fork, such as C 128. Bone-conduction will be unaffected, and the limit for lower tone hearing but little reduced. Let me repeat that this is the time to save the case, and add that, once the cause is removed, the ear condition will frequently recover without further treatment.

In the next stage, that of the organisation of inflammatory products, Eustachian obstruction is more marked, and the process will very likely have reached the tympanum, as shown by changes in the membrane beyond those of mere indrawing. There may be more or less impairment of the mobility of the malleus, and the tuning-fork reactions will show a greater departure from the normal. Rinne will be more decidedly negative, and this may be so for forks higher in the scale, such as C 512. Bone-conduction will be increased, and the low-tone limit still further involved. Even now, the removal of the cause and the restoration of the nasal air-way will bring about great improvement in the hearing, although it may be necessary to treat the ears in addition after operation.

It is when we come to the third and fourth stages that prognosis becomes more doubtful or decidedly less hopeful. It is in these stages that the symptom of paracusis Willisii, or hearing better in a noise, usually makes its appearance, a condition generally considered as a sign of great import as regards prognosis. It would be out of place to discuss here the cause of this symptom, but I may say that I do not agree with the doctrine that suggests it to be due to a flaccid tympanic membrane. I believe it to be due rather to fixation of the stapes, and I have as yet seen no case in which its presence was not accompanied by a negative reaction to Gellé's test. It is important to ascertain whether the membrane is stretched, because it is a valuable guide in prognosis and treatment. One important point, however, I would bring prominently to your notice. If these cases of flaccid membrane be examined carefully with the Siegle pneumatic speculum, in the great majority —if not all of them—it will be found *that the malleus is fixed.* Whether this fixation of the malleus, by preventing the movement of the membrane as a whole, is the real cause of the stretching of the posterior segment, which thus comes to bear the whole brunt of any increased intra-tympanic pressure, is the question. I would not like to say dogmatically that it is so, but so far as my present experience goes, I believe it is the true explanation. If this surmise is right, it would surely follow that any effort to reduce the flaccidity by artificial attempts to thicken and contract it cannot do good, save in preventing the flaccid portion from becoming sufficiently great to cause it to lie on the inner tympanic wall and embarrass the ossicular chain, and I very much doubt the possibility of achieving this. The opinion that I hold is supported by the fact that one sometimes sees cases in which atrophy of the membrane with a moderate amount of flaccidity, but with a mobile malleus, is not accompanied by marked deafness.

The chief points to be noted, therefore, in the diagnosis in cases of the last two stages of catarrhal middle-ear deafness are fixation of the malleus and fixation of the stapes. The presence or absence of these points makes a great difference in prognosis. In such cases the loss for low tones is greater, Rinne's test is doubly negative, that is to say, for high and low forks, and Gellé's test, when the stapes is fixed, is negative.

In some cases one meets with a chronic, slowly progressing middle-ear catarrh in which a chronic dry pharyngitis and naso-pharyngitis, without much or any history of frequent colds, has slowly extended to the tympanum through the Eustachian tube without causing any appreciable narrowing of the latter canal. They present all the changes met with in the later stages of ordinary middle-ear catarrh, and are sometimes called by the name "chronic dry catarrh." They are more difficult to manage in treatment, prognosis is not good, and they must be reckoned with in making a diagnosis.

A few words must be said in regard to the diagnosis between the late stages of catarrhal middle-ear disease and the condition known as otosclerosis. Some observers have confounded the two,

and error is likely to occur. The term "otosclerosis" should be confined rigidly to those cases to which the name—a bad one, but one that must be retained now for convenience—was originally given. Cases of late middle-ear catarrh, to which the term "otosclerosis" has been applied, should be considered rather under the title of post-catarrhal deafness. They show a marked degree of loss of hearing, and give the tuning-fork reactions for middle-ear deafness which have already been mentioned. The changes in the membrane and Eustachian tubes are such as have been described, and the cause will be demonstrable in the nose and naso-pharynx. True otosclerosis should be taken to mean a group of cases in which the nose and naso-pharynx are normal or present no special abnormality, the Eustachian tubes are patent, the tympanic membranes are normal in texture and inclination. A marked salmon-pink reflex is usually to be noted shining through the membrane in the region of the promontory or of the stapes, but may be absent, and the tests show fixation of the stapes, with a negative Rinne, a negative Gellé, increased bone-conduction, and marked reduction of hearing for low tones. Paracusis Willisii is a constant and early symptom. These cases are not middle-ear cases at all; they are due to a condition affecting the bony labyrinthine capsule, in which new bone is thrown out, ankylosing the footplate of the stapes in the fenestra ovalis, and osteoporosis occurs in the bony labyrinthine walls. With these changes is also frequently found degeneration of the specialised epithelium of the cochlea, so that, in some cases, there is loss of bone-conduction and reduction in the limit of hearing for high tones. These cases occur much more frequently in women than in men, begin usually between the ages of twenty and thirty, often start with pregnancy, and in a considerable percentage show a distinct heredity. They may be accompanied by ordinary catarrhal deafness, which makes the diagnosis more complicated.

In cases of catarrhal deafness in the later stages it is sometimes found that bone-conduction, instead of being increased, is lessened. If the diminution is not more than ten seconds it may be discarded, roughly speaking, but when it is over ten seconds careful testing for high tones should be carried out. The suggested implication of the internal ear in this class of case may be due either to true secondary internal ear deafness, to fatigue, or to want of exercise of the auditory nerve apparatus, or to the onset of senile deafness. In the first of these it is probable that an extension of the middle-ear condition occurs through the membrane of the round window, in which the loss of bone-conduction will amount to some twelve or twenty seconds, or even more, and will be accompanied by reduction in the hearing for high tones. This condition is especially liable to occur in the "dry catarrh" just alluded to. When the diminished bone-conduction is accounted for by fatigue or want of exercise of the auditory nerve (following want of adequate stimulation), the loss will not be great, and hearing for high tones will be good. Fatigue of the nerve may occur from the continued strain of trying to hear. A moderate loss of bone-conduction without corresponding loss of high tone hearing may be due to toxæmia, and I must repeat here the warning, expressed in the earlier portion of my lecture, never to overlook the possibility of a toxæmia, very often due to intestinal stasis, in cases of catarrhal deafness.

Senile deafness may appear earlier than is usual in middle-ear cases of some duration. It is important, when it occurs, to recognise this fact, and to avoid too active treatment. It is, unfortunately, a lesson which is only to be learned by experience that, in old people in whom senile deafness complicates old middle-ear catarrh, the former increases more rapidly than when it is the only condition present, and that, if too active treatment by inflation, etc., be tried in the hope of benefiting the middle-ear condition, the senile deafness is markedly increased.

One other word concerning senile deafness, or presbyacusis as it has been termed. I believe that, in quite an appreciable number of cases, the condition is determined by continued intestinal toxæmia. I am unaware whether this suggestion has been made before, but I believe it to be true.

I am reminded by the flight of time that we must proceed now to the consideration of certain points in connection with diseases of the internal ear before coming to a conclusion. And first let me touch upon injuries of this region. I have hitherto made no mention of such injuries as rupture of the tympanic membrane, but I shall speak of it here, because an important point in dealing

with a ruptured drumhead is to ascertain whether it is complicated with injury to the internal ear. An uncomplicated rupture of the membrane will usually heal readily and with good hearing, but, if there be any concomitant concussion of the labyrinth, as shown by loss of bone-conduction and reduction in the limit for high tones, the prognosis is much less favourable. Hence it is highly important to test every case with care if one is to avoid serious error in diagnosis and prognosis, for, be it remembered, some of these injuries may become the subjects of litigation.

Quite a moderate amount of violence may cause labyrinthine concussion, even without a ruptured membrane. It takes a considerable force to rupture a healthy membrane, and, in the majority of cases, there is some pre-existing disease when it occurs. Hence, occasionally, the force of a blow may be expended upon the labyrinth after passing through the more yielding membrane and ossicles. I remember a young man who, "larking" with his fellow clerks, received a box on the ear given in play with but moderate force. He became temporarily giddy, and, on recovering therefrom, was deaf in the ear upon which the blow fell, and complained of tinnitus. The tuning-fork tests showed this to be due to involvement of the internal ear and the deafness was permanent. No rupture of the drum occurred in this instance.

Another point upon which advice is sometimes sought is as to prognosis after injury to the labyrinth either by concussion or by a fracture of the base of the skull passing through the petrous portion of the temporal bone. Prognosis should always be very guarded in such cases, and it may be taken as a rough general guide that, if there be no improvement when six months has elapsed from the time of the injury, any recovery of hearing is hopeless.

There are many points in internal ear conditions that might be taken in this lecture, but time is too short to admit of our discussing them all. I shall, therefore, select a few of the more important and practical ones, and these can at best receive little more than mere mention.

Whenever a patient complains of vertigo he should be carefully examined by means of the labyrinthine tests to which reference has been already made. When chronic suppuration exists, examination should be exhaustive in order that a correct diagnosis as to the presence or absence of definite labyrinthine disease may be made. Sometimes vertigo is complained of in the course of a chronic suppuration which is due simply to the irritation of the labyrinth caused by the long-continued congestion of the middle ear or by pressure of retained discharge, cholesteatomata, polypi and the like, without the labyrinth itself being involved. These cases clear up readily under appropriate treatment, and it is highly important to differentiate them from the point of view both of prognosis and management. As a matter of fact every case of chronic suppuration of the middle ear that has lasted for some time should be examined to discover the exact condition of the labyrinth.

When vertigo occurs without suppuration, tests and history should receive careful attention. This cannot be entered upon here, but I would remind you that the symptom may, in some cases, depend upon a toxæmia.

In children a very large amount of severe deafness leading to deaf-mutism is caused by meningitis. We require to know much more about meningitis than we do at present. It is unfortunate that at present only the epidemic cerebro-spinal and tuberculous forms are notifiable, for, although notification of these diseases was based originally upon their infectivity, it is more than probable that numerous other forms of meningitis are due to infection. The only possible way in which we can cope with child-deafness from meningitis is by prevention, and until all forms of the disease have been elucidated by research we are powerless to effect much.

A rare form of internal ear deafness, when we consider the frequent occurrence of the disease from which it may result, is that due to mumps. It is a form which every practitioner should be well alive to, for it is only by its prompt recognition and immediate treatment that the hearing can be saved, and whenever a case occurs it is the imperative duty of those in charge to see that immediate measures are taken.

Lastly, in all cases of internal ear deafness in which there is any doubt as to the exact nature of the cause, search should be made for syphilis and the Wassermann reaction taken. I feel that, in speaking of syphilitic deafness, I should be neglecting my duty if I did not draw your attention to the brilliant research which we owe to my friend

and fellow worker in the cause of the deaf child, Dr. Kerr Love, of Glasgow. His work on the subject has but recently been published and should be known to every practitioner. If the coming Royal Commission on syphilis does its work rightly and fearlessly, we are on the eve of great improvements in our methods of dealing not only with the disease from its general aspects but as it affects the child.

I have just been speaking of the deaf child, and there is one matter which I must mention before I conclude. That is, do not make the mistake of confounding the deaf child with the mentally defective. This is a danger which the National Bureau for Promoting the General Welfare of the Deaf has tried to avert by safeguarding the deaf child's interests in the Mental Deficiency Act when before Parliament. If there is any doubt in the case of any given child that may come before you as to his being deaf or mentally defective, give him the benefit of the doubt, and let him have the chance of adequate special education for the deaf before labelling him as a mental defective.

I have endeavoured to deal with the rather large subject of errors of diagnosis in diseases of the ear from the points of view suggested to me by a somewhat wide experience, and I may, perhaps, have been rather too discursive. I feel that I have left out much that I might have said, but I have tried to offer for your guidance what occurred to me as of most practical value to my hearers. I have made no mention of the intracranial complications of middle-ear suppuration, of deafness from intra-cranial tumours and the like. These, however, are adequately dealt with in any good text-book on diseases of the ear, and once their discussion is embarked upon it is not easy to know when to stop. In conclusion, I would once again endeavour to impress upon you the great importance of care and exhaustive examination, and of looking at your cases comprehensively, not merely as a pair of ears, but as a complicated organism.

December 15th, 1913.

ON CERTAIN CONSIDERATIONS WHICH INFLUENCE THE QUESTION OF OPERATION IN OPHTHALMIC SURGERY.

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THE maxim *primum non nocere* is nowhere more important than in ophthalmic surgery. While eye operations seldom involve questions of life and death—malignant disease is relatively rare—much damage may be done by a very small intervention. The operative field cannot be sterilised, and at the same time the ocular media form a suitable nidus for the growth of organisms. Man, provided with two eyes, can as a rule get along very well with one, but loss of the second eye, which may be caused by injury to and even by well-considered operation on its fellow, is a misfortune of the worst kind. No eyeball should be opened unnecessarily and without a full sense of responsibility on the part of the surgeon. To be able to say, "Let well alone; I dare not operate," presupposes a great deal of bitter experience, since only by experience is it possible fully to realise how a comparatively useful eye may be rendered useless by an ill-judged or ill-timed operation.

Urgency in eye work is usually somewhat different in meaning from urgency in general surgery. In the latter it may be a matter of life and death whether an abdomen is opened or not, whether a strangulated hernia is operated on or not, but, except rarely, urgency in eye work refers to the preservation of sight, the relief of pain, or both of these, and not to the preservation of life.

GENERAL CONSIDERATIONS WHICH INFLUENCE THE QUESTION OF OPERATION.

Three considerations stand out rather prominently: The general health, the age of the patient, and the number of effective eyes which he possesses.

It is axiomatic that the best time to operate is when the patient is in good health. Such a choice of time is often not vouchsafed to us. When the general health is below par and delay not contra-indicated on technical grounds the

patient should be brought into as good health as may be before operation. This applies with greatest force to elderly people, in whom the state of the general health in relation to age, the mental balance, habits of diet, capacity for sleep, the urinary functions, bronchitic cough and rheumatism should all be taken into consideration where rest in bed for some time with one or both eyes bandaged is in contemplation.

Apart from general diseases, such as bronchitis and nephritis, in the presence of which the ophthalmic surgeon is reluctant to operate, the possibility of septic foci in any part of the body should be remembered. Conjunctival, lachrymal and palpebral sepsis are under the immediate eye of the ophthalmic surgeon, and oro-nasal sepsis ought to be looked for by him, but the general practitioner may well be on his guard against foci in other parts of the body. While a distant septic focus may or may not contra-indicate operation, it is right to bear in mind that it may be a possible source of infection, endogenous or exogenous.

Operations which are not urgent in the sense of being demanded for the preservation of life, however urgent they may be and often are from the standpoint of visual efficiency, must be regarded somewhat differently in the young and in the old. Leaving out of account the restlessness of children on the one hand and their more resilient tissues on the other, they have their lives before them. Thus it is urgently necessary to operate early on certain juvenile cataracts. The old have their days of usefulness behind them and often are content with very little. Instinct often tells them that it is wise to refuse to take the risk of failure, with its attendant consequence of a prolonged stay in bed.

But years are not everything. Contrast the frail and fretful, broken-down, possibly alcoholic individual not much past middle life, and the sturdy, optimistic and phlegmatic veteran of seventy or more. The latter class gives the surgeon every chance of success. It is true that the former sometimes do astonishingly well, yet in those whose health is poor for their age one ought carefully to weigh up the probabilities of failure before recommending operations. If operation has been decided on, then whenever possible it should be done under the most favourable conditions. Thus if the operation be one for the extraction of cataract and the patient be bronchitic the summer season should be selected. If the patient be dyspeptic he should be dieted and got up to his best. The question of alcohol is most important. No one willingly operates on a drunkard, but plenty of people take a certain amount of alcohol without being drunkards. Careful inquiry should be made beforehand as to the usual amount of alcohol, for it frequently happens that such an individual will be a much better patient if a certain amount of stimulant be given after the operation and during the following days than if his alcohol be cut off. This applies specially to the old person. The surgeon and the practitioner may uphold the principles of total abstinence, but discretion is the better part of valour.

Eye operations have a strong tendency to upset the mental balance, especially after middle life. No doubt alcoholism may be at the bottom of the matter in many cases, but by no means in all. A person who has never been addicted to drink may overstep the line for the first time, and even become maniacal, when put to bed after an eye operation with both eyes bandaged. It is a possibility that must always be reckoned with and as far as possible guarded against. If there is any suspicion that mental excitement is likely it is well to avoid double bandaging. If it does occur after operation the un-operated eye, sometimes even the operated eye, will have to be released. A great deal can be done with sedatives from the standpoint of prophylaxis and of treatment. Forced abstention from alcohol, certainly, and from tobacco probably in those who are accustomed to these as sedatives, may be contributory causes of mental derangement. One must remember that in many eye operations the general health is normal, and therefore the capacity for enjoyment of usual luxuries is but little impaired. One should make as little change in the diet as is compatible with the recumbent position and the maintenance of sufficient quiescence of the muscles of mastication. The judicious exhibition of a little stimulant at the psychological moment may save a patient from post-operative mental disturbance, by contributing to his restfulness of mind. I do not know whether any surgeon allows his patient to smoke immediately after an eye operation—it is hardly probable—but it is certain that patients do all manner of wrong things on their own initiative without any particular harm accruing. No doubt they sometimes smoke

without the surgeon's knowledge. Many continental surgeons, after balancing up the advantages and disadvantages, have come to the conclusion that restlessness is such a serious factor in post-operative treatment that they allow their patients to sit up in a chair the day following a cataract extraction. That British surgeons have not seen their way to do this as routine practice is probably due to the fact that British nursing is so much superior to that usually to be obtained on the continent. Still, the fact remains that no harm seems to follow the practice.

As to the administration of general anæsthetics to the old, the general practitioner and the trained anæsthetist may be left to decide the question. One ought to distinguish between the meaning of the word "urgent" when applied on the one hand to an operation demanded for the preservation of life, and on the other hand applied to an operation for the preservation of sight, for the relief of pain, or for the improvement of appearance, which cannot be undertaken under local anæsthesia without causing pain or shock or both. In the old it may at times be better to temporise even to the risk of loss of sight, or to allow the patient to suffer pain up to a certain point, than to take the risk of general anæsthesia.

The third general consideration which influences the question of operation in ophthalmic work is that as to the number of effective eyes possessed by the patient. What is our duty to the individual with one eye, or with only one seeing eye? While not forgetting the value of the binocular field and of stereoscopic vision, the great value of the duplication of the eye is to provide against the contingency of injury. A person may lose one eye not only with comparative equanimity, but often also with but little reduction of working ability. (Wage-earning capacity is another question, thanks to the law.) Binocular vision, that is to say, is *relatively* a small matter. If the second eye be lost the individual is apt to become useless to himself and to the community. In the one-eyed individual no stone must be left unturned in seeking to cope with advancing disease, but operative treatment must be weighed and considered. Among the possibilities of failure there looms large in all cases the risk of sepsis. No eye operation, however apparently safe, can escape the risk. It may be greater or smaller, but is always present. In eye operations wound infection nearly always spells failure, and considerable or complete loss of the function of the eye. In considering these very perplexing cases, no matter how temptingly probable a good result may be, one should always ask—"What if the eye be lost?" Personally I will hardly be persuaded to open the eye of a one-eyed individual unless sure of one or other, or both, of two things: (1) that the eye as it stands is visually useless and without prospect of spontaneous improvement, (2) that there exists a progressive disease, leading to blindness, which has a chance of being stopped by operation. From this net of circumstances there will escape a class of case in which there is sight enough to get about with, and in which the disease is either non-progressive or only very slowly progressive. Rejecting as too speculative all plausible arguments as to possibilities of a brilliant result, such eyes should in my view be considered as crying "*Noli me tangere.*" I have spoken only of sepsis, but it is not the only road to ruin in eyes which are already diseased.

I will now consider the question of operation in a very few individual eye diseases.

Cataract.

Cataract is frequently the only ascertainable deviation from the normal present in an eye. Hence, the brilliance of the results which are obtainable by operation. Yet it is this very brilliance which throws into relief the occasional failures which occur. The circumstances influencing the question of operation to which I wish to refer are three, chosen specially because they may appeal to the general practitioner and help him to explain certain facts to his patient.

(A) *Unilaterality of cataract.*—Frequently one has to decide upon the course to be adopted when a cataract is present in one eye only, the other being free. Such patients may have a considerable span of life before them. I do not refer now to juvenile cataract or to traumatic cataract. The reason for the onset or progress of cataract in one eye is often obscure, though there are cases in which some small defect in the lens of the other eye makes one suspect a congenital origin. Sometimes the cataract is associated with myopia or has followed other disease of the ocular tunics. The prognosis may be seriously affected by such origin, but we

shall suppose for the present that the light projection of the affected eye is good, and that there is no reason to expect that there will be serious technical difficulties. Reduced, then, to the terms in which such cases really require much consideration, we have here a unilateral obstruction to vision, and there is every prospect that removal of the obstruction can be successfully accomplished and vision restored. Then why not operate at once? Unfortunately the question of operation is not to be so easily settled. Suppose the result is technically perfect, and that an eye previously approximately emmetropic has obtained good vision aided by a strong convex glass. This strong convex glass cannot be worn while the other eye wears none. Let us see what the patient has gained and what he has lost by the operation.

(1) He has regained the full field of vision. He can see objects moving on his previously blind side. That is a valuable gain in certain occupations. In other occupations it is of little moment. It is a great gain to anyone in a crowded street.

(2) He is in a strong position should anything go amiss with the fellow eye.

(3) He is relieved of anxiety.

(4) He has (assuming the fact) had his operation performed at the favourable moment when his cataract was "ripe," but not "over-ripe."

What about the loss?

(1) He has, especially if he be a highly strung and well-educated and observant individual, lost the clearness of vision associated with the use of one good eye, and has had substituted for it a certain amount of confusion, possibly diplopia, caused by dissimilar retinal images.

(2) Worse still, unless the position has been made as clear as daylight beforehand by the most careful explanations, he may have lost confidence in the surgeon who has failed to give him an eye as good as the other one. No amount of subsequent explanation will ever persuade a certain class of patient that the result obtained is the very best possible under the circumstances.

It is mainly this second "loss" which has determined my personal practice in these unilateral cases. In such cases in adults it is my usual rule not to urge or even to admit the advisability of operation so long as the cataract is in the very least immature. Probably when the cataract is fully mature it is best to operate without waiting any longer, and in any case it should not be allowed to pass into the over-ripe stage. Hypermature cataracts are apt to give rise to complications in their treatment, and when the fact is put before the patient that further postponement is risky, on technical grounds, he can at the same time safely be told that the practical visual gain to him at the moment is not the main consideration. So long as the cataract is immature the surgeon cannot truthfully put forward the safety of the eye as his reason for operating, but must rely, so far as satisfying the patient is concerned—and that is my point here—on the actual visual benefit to accrue. Such visual benefit is often a very doubtful asset in the patient's subsequent opinion, and unless he fairly understands the value of a full visual field he is very apt to be disappointed in what he has received.

(B) *Bilateral immature cataract.*—Next, as to the patient who has two cataracts, neither of them mature, and who has come to the end of his working ability. These cases are often difficult to decide upon, especially when the patient is comparatively young, and when the lenses are not likely to be fully sclerosed. In old people there need not, as a rule, be much hesitation in operating at once on the worse eye, because the lens will probably deliver pretty cleanly even though the opacity be very incomplete. In patients in their forties and fifties it is otherwise. The lens cortex is apt to be sticky and to remain in part *in situ*, giving rise to secondary cataract and other troubles. Here the operation of extraction of the cataract in the capsule should have its principal field of usefulness, but up to the moment this procedure is not in favour with many European surgeons. Some surgeons are in favour of operating freely on these cases, washing out the cortical remains from the anterior chamber by means of some form of irrigator. Probably the majority are not in favour of this, at any rate as routine. My own preference is to perform iridectomy upon the worse eye, sometimes massaging the lens through the cornea very lightly. Frequently the cataract thereafter passes more or less rapidly towards maturity and can be extracted in the usual way. If it does not, and if the patient's condition urgently demands that something should be done, the immature cataract must be extracted in whatever manner may seem best.

The boldness of some operators who operate

upon both eyes at the same sitting does not appeal to many surgeons in this country.

(c) *Juvenile congenital cataract.*—Under this heading I intend to speak only of the treatment of cataract in young children in so far as the treatment is influenced by their youth. I wish to speak of the slow *versus* the rapid method of dealing with these cataracts. By many operators congenital cataract in young children is treated by the rapid method. This consists in freely needling the cataract, allowing the lens matter to swell quickly and then drawing it off by means of a keratome incision. Some congenital cataracts are quite fluid, and when needled the anterior chamber becomes filled with a viscid milky material which generally requires to be drawn off. I am not thinking of this kind of cataract just now but rather of the usual lamellar type.

With regard to the rapid method just mentioned, Herbert Parsons has expressed the opinion, speaking especially as a pathologist, that the rare cases of cataract operated upon in childhood which do go wrong are precisely those which have been treated by this method. On clinical grounds and from my own observation I believe that Parsons is right. Firstly, unless the operator can be continually in attendance, or can trust absolutely to the general practititioner or the nurse to give him warning, glaucomatous symptoms may arise and be neglected. As Sym has insisted upon in his text-book, vomiting is in these cases an important sign of rise of ocular tension. Doubtless such glaucoma is not common, but it does happen very rapidly sometimes, and the tension may rise to a high degree in a few hours. Secondly, the iris may become caught in the wound when the lens is drawn off by the keratome. Thirdly, the vitreous may present in the wound at the time of the keratome operation and give rise to complications. Fourthly, it may be necessary to perform an iridectomy to prevent the occurrence of No. 2. While I am a strong upholder of iridectomy in the extraction of senile cataract I think it is quite unnecessary in children, and, from the point of view of appearance all through life, decidedly to be avoided if possible. I prefer the slow method. It may take a long time from the first operation on the first eye to the last on the second eye, but, considered in regard to the security offered, what matters the loss of time to a child? Some will argue that the method submits the patient to the repeated risk of sepsis. The risk must be admitted. But in all probability it is smaller for a number of needlings performed by a careful surgeon than for one linear extraction. Nevertheless, the fewer needlings the better. One needling, and patience, will often go a long way towards lens absorption.

Since one can never gauge the amount of swelling in any given case my own practice is merely to scratch the capsule at the first operation. Probably there is very little swelling. A week later the capsule is opened more freely but not by any means very freely. If too much has not been done the lens swells slowly and is gradually absorbed in the course of some months. Another needling or more than one may be necessary if the process tends to stop altogether, and a final division of the capsular remains may be required in order to clear the pupil. As a rule I allow one eye to have useful vision before beginning on the other. The pupil must be kept widely dilated all the time and the possibility of rise of tension remembered. The general practitioner can be of much assistance in these cases. The intervals between operations may be considerable and the patient some distance from the ophthalmic surgeon. The family practitioner, by keeping a constant watch for the symptoms of irritation (contraction of the pupil, lachrymation, redness of the eye) or of glaucoma, which now and again come on unexpectedly in an eye which has up to the moment remained quiet, can send the patient to the ophthalmic surgeon before much harm has been done. Taken all over, this slow method is to my mind the better one, especially, perhaps, in quite young children, since time is of no particular moment, and because the after-treatment of needling is easier than when the eye has been opened by a keratome incision. A restless intractable child is less apt to do harm to an eye which has had the needle operation only than to one opened by the knife.

High Myopia.

Of all the operative procedures which may be adopted in eye diseases there is hardly one in which the maxim *primum non nocere* has greater force than in the *operation for high myopia*. Not that a brilliant success may not often reward the surgeon, yet on the other hand the result of needling the clear lens in this disease may be disastrous.

The risk is twofold: (1) Sepsis, which is common to all operations; (2) subsequent loss of sight through detachment of the retina, glaucoma, or other complications which are prone to occur in any eye with such a degree of myopia that operation comes within the range of practical politics. The risk of sepsis may be small, but the risk of disturbing a diseased vitreous and uveal tract is a constant one, not only in myopia but also in any eye in which these parts are affected.

The operation is theoretically a tempting one. A myopic eye is to be rendered approximately emmetropic. The myopia is to be got rid of. But the thing is not really so simple. Apart from the fact that accommodation is abolished by the operation the myopic eye is not a normal organ, and may react in such a way as to become of less use than before. It may become blind instead of merely bad-sighted.

Apart from the question of the precise degree of myopia which calls for operation, all are agreed that the operation is best performed in youth and only when the fundus is comparatively healthy. In my opinion the operation is practically always too risky. To operate on both eyes is quite unjustifiable. The risk to sight of this method of treating high myopia is a "condition" or "circumstance" influencing the question of operation which I have thought might be usefully included in this article, although, indeed, the operation is not very often done.

We have seen that *retinal detachment* may follow the operation for high myopia. It frequently occurs in myopia which has not been so treated, and it may be an argument in favour of operation in myopia that detachment of the retina is apt in any case to come on spontaneously. True, it may be said, detachment sometimes follows the operation, but then it would probably have developed in any case. There is all the difference between the two cases from the standpoint of the surgeon's responsibility. In the one case he is likely to be blamed for the detachment, in the other he cannot be blamed for it.

But the operative treatment of *spontaneous detachment* occurring in myopia is another matter. Here we are dealing with one of the most obstinately incurable of eye diseases. Shall we attempt to cure by operation or not? This is a very interesting question which I have already gone into at considerable length in the columns of the 'Ophthalmoscope' in 1910. I endeavoured to work out from a close study of the literature the proportion of cures by operation as compared with other methods.

"This figure, 10 per cent., which represents cures by various operative methods, does not take any account of the negative results of some writers, and does not convey any hint of the important fact that, as the result of some operations, patients have been made worse. If alongside this 10 per cent. we place the more optimistic of the figures for cures of a spontaneous nature, cures which have come about by doing nothing, or by simple rest, atropine, attention to the bowels and diaphoresis, we get 7·2 per cent. Admittedly this is a high figure; but when we remember that we run no risk of making the patient worse by our own efforts, even a lower figure would not leave a very great *direct* advantage—for operative over 'pacific' treatment. But there is another point in favour of operative treatment. It must be allowed to have this *indirect* advantage—that it creates confidence in the patient that something is being done to alleviate his pitiful condition. Spontaneous cures are sufficiently numerous to allow us to leave the decision to the patient without too much pressing forward of an operation, but if he leaves the matter to us, should we not offer to operate in recent cases where a fair trial has been given to rest in bed, etc., and in old cases where there is any possibility of success?" We are dealing with a condition in which the eye is well-nigh worthless. It *may* recover spontaneously—very few do. It may recover as a result of operation—very few do. If the patient wants an operation, I do not think that either the surgeon or the patient stands to lose anything in the event of failure to improve the sight.

Strabismus.

It is the fashion at the present day to operate in cases of squint by advancement to the exclusion of tenotomy either alone or combined with advancement. Now I am far from denying that a skilfully performed and properly managed advancement on one or both eyes is the best operation for convergent concomitant strabismus and is the only operation in divergent strabismus. But circumstances occur more frequently than is generally supposed in which advancement may give place to the older and unfashionable method of tenotomy to the

advantage of the patient. Besides, if a complete tenotomy be not considered right there are various methods of lengthening the tendon.

What does an advancement mean to the patient as compared with a tenotomy? Most operators will agree that for an advancement to be successful the patient must have both eyes bandaged for a week or the better part of a week. After that he will be incapacitated for work for another week or two. If the one operation is insufficient, as may be the case, then the patient must undergo an advancement operation on the other eye, so that more time is lost. It is true that some operators will advance both recti at once, but this plan is not generally in favour.

From this point of view, then—the loss of time and the expense involved in the loss of time—advancement is a rather serious proposition for the working man or woman. Do we always, or even generally, gain by advancement such an advantage in the result as will compensate for all this? I certainly do not think so. We may upon occasion obtain everything for which we wished—binocular simultaneous vision and correction of the deformity. More often the latter only is obtained, or partially obtained. In adults, indeed, it is frequently the case that the original visual acuity of the squinting eye is such that binocular vision could not be expected. Granting that correction of the deformity is the main point in the opinion of the adult patient —and this is certainly the case in most instances—is the cosmetic result of tenotomy alone likely to be as good as that of the more elaborate operation? That is a question which for various reasons does not admit of a categorical answer. It is true that a badly performed tenotomy may indeed give a very poor cosmetic result, and that tenotomy should not be undertaken, especially in children, without a full study of the case, but in adults who can ill afford the time or the money for lying up, a simple tenotomy, carefully performed, which requires no confinement to bed, which can be carried out between a Saturday and a Monday, and after which a patient can return to work in a few days, often will give a cosmetic result which completely satisfies the patient and costs him but little. Further, if we look at the other side of the shield and contemplate the possibility of a convergent squint being converted into a markedly divergent one as the result of tenotomy, I think it must be admitted that the risk is not a great one provided the operation is performed with skill. In the twenty years of my experience, during which time many cases tenotomised by my predecessors have come under observation, I have but rarely seen divergence as a result, and in some of these the tenotomy had undoubtedly been too freely performed. Besides, a moderately performed tenotomy should not result in loss of the tendon within the orbit. The operation for its advancement may not be easy, but even years afterwards it may be possible to bring forward the tendon again.

I wish, then, while not discussing the kind of cases which are suitable or unsuitable, to say to the general practitioner that advancement is not necessary in every case of convergent squint. The circumstances of the patient, visually and financially, should often influence the operator to discard scientific ideals, and to do something for the patient which will satisfy reasonable requirements without putting him to too great an expenditure of time, of money, or of both time and money.

This article might have covered many other subjects, but my object has merely been to discuss with the general practitioner some points of view regarding eye operations which may be of service to his patient. To speak to a non-specialist on such highly technical points as the treatment of glaucoma or what is best to be done in penetrating injuries of the globe with risk of sympathetic ophthalmitis would lead to nothing, because in my opinion such disorders are essentially those in which from the point of view of the good of the patient there is no room for any but the opinion of the specialist. It may be open to question whether a certain person should or should not have a cataract extracted, and the discussion may be taken part in to the good of the patient by the patient and by the practitioner, but in such a case as that instanced where it is a question as to the advisability of enucleation of an eye for actual or anticipated sympathetic ophthalmitis, no opinion except that of the specialist is of any value at all. If after a long special experience any man can persuade himself that he has not made many mistakes in dealing with such subjects, that man is to be envied his peaceful mind and his optimistic temperament. It is just the experience of disasters in his own practice and in the practice of his colleagues which gives so much weight to the considered opinion of the specialist as to what is best to be done in really difficult cases. Therefore, in speaking about individual diseases I have been able to select only one or two of those in which there is room for discussion between the patient, the practitioner and the specialist.

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COMMON ERRORS IN THE DIAGNOSIS OF THE INFECTIOUS DISEASES.*

By E. W. GOODALL, M.D.,
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THE text of my address is to be found in a certain statistical table which is published every year in the Medical Supplement of the 'Report of the Metropolitan Asylums Board.' This volume is, I fear, known only to a few, and chiefly to those who are engaged or interested in the management of fever hospitals. Yet it contains a large amount of information which is useful not only to the class of persons I have just mentioned, but also to the statistician, the London ratepayer, and the medical practitioner. The table to which I refer is that which gives details of the miscellaneous diseases admitted to the Board's fever hospitals. As you are doubtless aware, no patient is received into those hospitals unless he is certified by a medical man to be suffering from one or another of some dozen acute infectious diseases, for the treatment of which accommodation is provided by the Board. The miscellaneous diseases enumerated in the table are diseases which have been sent to the hospitals in mistake for one of these infectious diseases, and a perusal of the list affords food for reflection. If we consult the volume last issued, that for the year 1912, we shall find that at least 1856 mistakes in the diagnosis of the acute infectious diseases were made in London during the year in question—that is, 8 per cent. of the total number of admissions, which was 23,301. As a matter of fact the table does not represent the whole of the error; for it does not show the number of cases in which one admissible infectious disease has been mistaken for another—for instance, scarlet fever for diphtheria. A case of scarlet fever which

* Lecture at the Polyclinic.

is sent to hospital as diphtheria is included in the tables devoted to scarlet fever. The percentage of error in diagnosis varies from year to year. Taking all circumstances into consideration I should be inclined to say that it is usually about 10 per cent.—that is to say, one case out of every ten which is sent to one of the Asylums Board's fever hospitals is not what it is certified to be.

Now, in my opinion, this proportion is too high. I fully acknowledge that in the case of some diseases, *e.g.* rubella and typhoid fever, the diagnosis is often difficult, that the circumstances in which the diagnosis must be made are at times most unfavourable, and that in some instances the retention of a suspicious case of infectious disease in its surroundings is highly undesirable. I say I freely acknowledge these pleas in mitigation, and yet am still of the opinion that the number of cases of misdiagnosis is too large.

It appears from the table in the Asylums Board Report for 1912 that there were, in round numbers, 100 different diseases which were mistaken for the five common acute infectious diseases which are admitted into the Board's hospitals, viz. scarlet fever, diphtheria, typhoid fever, measles, and whooping-cough. Many of these 100 diseases, however, are responsible for only one, two or three cases apiece; but there are certain diseases which stand out conspicuously as apparent sources of trouble. These are: (1) certain of the other acute exanthematous infectious diseases, especially rubella and chickenpox; (2) various cutaneous affections, usually acute; (3) various inflammatory diseases of the fauces and larynx; (4) bronchitis, broncho-pneumonia, and lobar pneumonia; (5) besides the diseases which are named in the table certain of the infectious diseases which are admissible to the Board's hospitals are mistaken the one for the other.

Now it will be quite impossible for me, in the time at my disposal, to discuss the differential diagnosis of all these diseases. I think I can most usefully employ the time in stating broadly what I believe to be the principal causes of these mistakes and in endeavouring to show how they can be avoided.

Anyone who looks over the table to which I have referred must almost involuntarily ask himself, How comes it about that these mistakes are made? An experience of many years leads me to believe—nay, I will go farther, and say leads me to be sure—that the causes of the errors are as follows:

(i) Difficulties due to the surroundings of the patient.

(ii) The necessity of arriving at a diagnosis on probabilities in order to obtain the early removal of a case that is possibly infectious.

(iii) Inexperience and ignorance.

(iv) Carelessness.

(v) Faulty methods of examination.

(vi) Inherent difficulties.

It is not necessary for me to enter into details concerning all these causes. By difficulties due to the surroundings of the patient I mean a poor light, a dirty condition of the patient's skin, and so forth. In my opinion, however, these conditions are not responsible for any large proportion of the errors. A very considerable number of the mistaken cases come from the out-patient departments of the general and children's hospitals of the Metropolis, and these causes are not in operation in these institutions. There the powerful twin factors, inexperience and ignorance, are at work. Nearly all the cases from these departments are certified by men who have just qualified, and have not had the opportunity of becoming at all intimately acquainted with even the common infectious diseases. Unfortunately, but still certainly, I see too many cases in which nothing but ignorance can have led to the diagnosis. While engaged in preparing this address, within three or four days three patients were sent to the hospital as cases of scarlet fever who were suffering from, respectively, the marginate variety of hyperæmic erythema, an enema rash, and peliosis rheumatica. Ignorance alone could have been the cause of the misdiagnosis, so different were these rashes from that of scarlet fever. Well, as I have said, many of the mistakes under this head are committed by the young and inexperienced, but by no means all. Not a few which come in this category are due to the prevalence of certain beliefs which are dying very hard. The chief of them are as follows:

(*a*) The belief that if a scarlatiniform rash is present the patient must necessarily be the subject of scarlet fever. Now a punctate erythema, having more or less the distribution of the rash of scarlet fever, may be seen—

(i) As an initial or prodromal rash in measles, chickenpox and smallpox.

(ii) As a phase of the rash proper in rubella, and occasionally in measles.

(iii) As the effect of a drug, such as belladonna and quinine.

(iv) As the result of an enema.

(v) In certain, fortunately not very common, skin-diseases, of which the most puzzling is recurrent scarlatiniform erythema.

(*b*) The similar belief that a morbilliform rash indicates measles and little else. Such a rash occurs—

(i) As an initial or prodromal rash in smallpox.

(ii) As a so-called secondary or accidental rash in scarlet fever, measles, diphtheria, typhoid fever, and other infectious diseases, including acute rheumatism.

(iii) As the effect of a drug, such as antitoxic serum and copaiba.

(iv) As the result of an enema.

(v) In connection with any septic condition. Under this head it may be mentioned that septic sore throat is not infrequently accompanied by a rash of this description. The secondary rashes mentioned above are closely allied to this form.

(vi) In certain skin diseases, especially erythema multiforme and the hyperæmic erythemas.

(*c*) The excessive importance attached to the condition known as a "strawberry tongue" as a sign of scarlet fever. This state of the organ is met with quite as often in measles as in scarlet fever; it may be seen in diphtheria, and is not at all uncommon in cases of sore throat accompanied by a septic rash, and even in any case of erythema, whatever its cause may be.

(*d*) The belief that if desquamation follows a rash the case must be one of scarlet fever, and the converse belief that if the rash is not followed by desquamation the disease cannot be scarlet fever.

These beliefs have been the cause of many mistakes. Any erythema and dermatitis may be followed by peeling. I have seen well-marked desquamation, quite indistinguishable from that of scarlet fever, after measles, rubella, a serum rash, and a septic rash. Peeling may also occur when there has been either no rash at all, or but a transient and trivial one, as, for example, in typhoid fever and acute rheumatism.

What I have termed "diagnosis on probabilities" is, in my experience, becoming a more frequent cause of misdiagnosis. In some instances the practitioner is placed in a dilemma. If he delays diagnosis till certainty is reached, the case may be left in most unfavourable circumstances, supposing that ultimately it turns out to be one of infection. On the other hand, if he makes a diagnosis on insufficient evidence, he runs a risk of having his diagnosis corrected later by the hospital or public health authorities. Naturally, few of us like to be thus put right. Simple laryngitis (of which 102 cases appear in the table) is a case in point. It is impossible without certain evidence to pronounce whether or not the patient is in the early stage of an attack of laryngeal diphtheria. Yet, if the case *is* one of diphtheria, delay is most dangerous, from the points of view both of the dissemination of the disease and of the treatment of the patient. There are circumstances which justify a diagnosis on probabilities, and even make it commendable.

As for the next two causes, carelessness and faulty methods of examination, I will take them together as they are akin. One variety of the former is the vice of making rapid diagnoses. I have known it lead to most disastrous results. In one instance an outbreak of smallpox in a hospital ward was due to the offhand diagnosis of a case of modified smallpox as one of acne varioliformis. In another, a fatal outbreak of measles, also in a hospital ward, was due to a rapid diagnosis of a prodromal rash of measles as scarlet fever. But most examples of these errors are, I think, due to faulty methods of examination. The observer has no system, consequently he accidentally omits to inspect a certain region of the body or to scrutinise certain details and thereby misses valuable information. I have found that usually such an observer is very hazy in his ideas of the various lesions met with in cutaneous diseases. For instance, he does not appreciate the difference between a papule and a macule, or is quite indifferent to the necessity of paying attention to the distribution of cutaneous lesions. I am inclined to attribute a large proportion of the diagnostic errors to these faults. For example, the disease most frequently mistaken for typhoid fever is acute pneumonia. Nearly all the cases of this disease present the physical signs of pulmonary consolidation more or less well marked when they arrive at the hospital, and the other symptoms which may happen to be present can in all but an insignificant minority of the cases be accounted for by the easily diagnosed lesion.

Acute pneumonia is not a common complication of typhoid fever, and when it does occur comes on late in the disease. But often a patient with acute pneumonia will complain of abdominal pain, the pain of pleurisy referred to the termination of the lower intercostal nerves; he may also suffer from diarrhœa. The abdominal pain, diarrhœa and pyrexia, combined, I am afraid, with an omission to auscultate the lungs thoroughly, lead to the erroneous diagnosis.

I offer no apology, even to my present audience, for drawing attention to these causes of mistakes. We are all liable to be caught napping, even those of us—I think I may say especially those of us—who are not young and inexperienced. The causes I have mentioned are very real sources of error in the diagnosis of the common acute infectious diseases, and some of them are causes which can and should be removed. I would especially like to see the end of the desquamation fetish.

Lastly, there are the difficulties inherent in the nature of the case. These may confound the most careful, conscientious and even experienced observer. Rubella and typhoid fever, for example, are notoriously puzzling, as also are anomalous cases of various acute infectious diseases. But I will confine my observation to the most common of these genuine difficulties—difficulties which are set up neither by the circumstances of the case nor by the attitude of the physician.

Rubella (rötheln, German measles) is a source of much trouble. Fortunately it is not constantly with us, nor are epidemics very frequent. I am not surprised that it is often mistaken for scarlet fever, for at one stage the appearance of the rash will deceive even the elect. In the vast majority of cases of this disease the eruption comes out first on the face in the guise of small, pink, discrete spots. They may be very faint. If you see these spots on the face, scarlet fever is absolutely excluded. The spots soon come out on the trunk. In the next stage the spots disappear from the face, and those on the trunk either become confluent to form, or are overwhelmed by, a diffuse erythema, which is often exactly like that of scarlet fever. This is the difficult stage of rubella; and there are not a few cases in which at this stage I should decline to make the differential diagnosis if I had not previously seen the eruption on the face myself, or had the information of its presence in that situation from some person upon whose powers of observation I could rely. But in some cases the rash is still spotty on the extremities, the spots being small and discrete. Often the superficial glands are enlarged. Still I must admit that to confuse rubella with very mild scarlet fever cannot always be avoided. As for the distinction between rubella and measles, in the former there is rarely a prodromal period and the rash is one of the earliest signs. In rubella the buccal mucous membrane is normal; in measles it rarely is. In measles even if Koplik's spots are not to be seen (and they usually vanish when the eruption is out), the interior of the mouth has a dry, finely granular and abnormally red appearance. In about three fourths of the rubella cases all the superficial glands are moderately enlarged; they are enlarged in but a small percentage of scarlet fever and measles cases. A marked faucial lesion excludes rubella.

I have already given a list of the diseases in which a punctate erythema may be seen. Amongst the infectious exanthemata besides scarlet fever and rubella the most common are measles, chicken-pox and smallpox, in which it occurs as an initial or prodromal rash. Never diagnose a case as one of scarlet fever without examining the mouth for Koplik's spots. Always remember that the cheeks and forehead and the area round the mouth and nose are never invaded by the proper rash in scarlet fever. The rash of scarlet fever is usually evenly distributed and not patchy. It is rarely bounded by an abrupt edge. It usually involves the limbs. It is, when fully out, punctate, and the more brilliant, the more it is punctate. Some of the scarlatiniform initial rashes of smallpox are most intense over the lower part of the abdomen and the upper and inner parts of the thighs. In fact this is a diagnostic distribution.

An enema of water and ordinary hard yellow soap will not infrequently produce a punctate erythema. It is usually confined to the trunk, and is unaccompanied by other symptoms, though in a convalescent typhoid patient I have known it to be accompanied by some pyrexia, and even a rigor. Occasionally the rash is macular, and then the face may be invaded.

The most common morbilliform rashes to be mistaken for measles are certain initial rashes of smallpox, the early stage of the proper rash of smallpox, the accidental or secondary rashes of scarlet fever, and other acute infectious diseases, septic and other hyperæmic erythemas, and erythema multiform. In the condition of the mouth, to which I have previously referred, will be found a sure guide to measles. There is also seldom wanting

in this disease catarrh, more or less extensive, of the respiratory tract, which gives rise to coughing and sneezing. These conditions are absent from smallpox.

Septic rashes are prone to be confined to the extremities, especially their extensor surfaces and the joints. If the face and trunk are invaded, the rash has appeared on the limbs first. These rashes are occasionally seen during the course of the acute infectious diseases, and are those which I have mentioned as "secondary" or "accidental" rashes. Serum or antitoxin rashes and many drug rashes belong to the same class, which is allied to the group of exudative erythemas. These rashes are frequently macular, papular, and marginate. It is worthy of note that the true measles rash is never marginate or ringed. If in a case of a doubtful macular rash any portion of it shows a ringed arrangement, caused by the fading of the centre of the macule as the edge spreads, then the case is not one of measles.

It is remarkable that urticaria is sometimes diagnosed as scarlet fever or measles. But though urticaria is quite unlike the rashes of these diseases, it must be remembered that it may occur as the prodromal rash of measles, and as a secondary rash in any of the acute infections.

In smallpox times, about half the cases which are misdiagnosed are cases of chickenpox. But the mistake can almost always be avoided by paying a strict attention to the distribution of the eruption in the first place, and the character of the pocks in the second. I cannot emphasise too strongly the wisdom of not arriving at a diagnosis of any case in which there is an exanthem without a survey of the whole of the patient's skin.

Of the misdiagnosis of laryngitis for diphtheria I have already spoken. I will only add that before you make the diagnosis of diphtheria (I am referring to cases in which there is no faucial evidence of the disease) always make certain that the case is not one of measles or of a retropharyngeal abscess, or of a foreign body in or about the larynx.

The only other common diseases over which errors are made are various acute lesions of the fauces, which are mistaken for diphtheria. In regard to these lack of time will not permit me to say more than that most of the cases which are not clinically diphtheria are also not bacteriologically diphtheria; that diphtheria seldom presents itself as a general inflammation of the fauces without the formation of a definite false membrane; and that ulceration and sloughing of the tonsils or fauces are very rare in diphtheria.

I will conclude by showing and explaining a few slides illustrative of some of the points about which I have been speaking.

December 22nd, 1913.

PESSARIES VERSUS OPERATIONS IN THE TREATMENT OF UTERINE DISPLACEMENTS.*

By ARTHUR E. GILES, M.D., B.Sc., F.R.C.S.,
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In former days displacements were not treated except by the means of pessaries; and the only question to exercise the mind of the practitioner related to the kind of pessary he was to use. Certainly this was a large question, if we may judge from the number and variety described in the text-books of the day. Most instruments and inventions when obsolete are forgotten altogether; but pessaries appear to be cherished by surgical instrument manufacturers, whose catalogues, when these appliances are concerned, read like a catologue of museum specimens.

In the last twenty years the ingenuity of gynæcologists has been diverted from the invention of new pessaries to the devising of operations for the relief of displacements. At the beginning of this surgical era the indications for the use of pessaries were numerous, and the indications for surgical intervention were very few. The progress of surgery in the direction of improved technique and diminished mortality has led to a steady encroachment of surgical procedures on the domain of pessaries, and it does not require a seer to foretell that in the course of time the original conditions will be reversed, and the indications for surgical treatment will be numerous, and the indications for pessaries will be few.

Of course, I am referring to the attitude of the profession as a whole; for just as, at any given time, there are always some ardent spirits whose ideas are in advance of their day, so there are always cautious and conservative people who lag a generation behind. In this, as in other matters, we find exemplified the proposition enunciated by the late James Hinton, that "truth lies in the polar union of opposites."

I wish now to define the relative scope of the

* A lecture delivered at the Polyclinic.

indications for pessaries and operations from the standpoint to which my own experience has led me. Without entering upon debatable questions of ætiology and pathological anatomy, I shall include under the term "displacements" retroversion and retroflexion of the uterus, prolapse of the uterus and vaginal walls, and procidentia. Moreover, since some backward displacements of the uterus cause no symptoms, and are, therefore, best left alone, I may add that my observations refer only to cases in which symptoms are present from which patients must be relieved.

From the point of view of treatment I recognise three groups of displacements:

(*a*) Cases in which there is a clear indication for pessary treatment.

(*b*) Cases in which there is a clear indication for operation.

(*c*) Optional cases, in which the patient should have both courses explained to her, and be allowed to choose.

Such divergence of opinion as is found on the subject has reference primarily to the position of the boundary lines between these groups.

(*a*) Cases in which there is a Clear Indication for Pessary Treatment.

In this group I should include the following cases:

(1) Retroversion of the uterus following confinement. In many of these cases there is a temporary relaxation of the ligaments and loss of tone, and the wearing of a Hodge pessary for a few months will usually bring about a cure.

Let me note in passing that when I speak of a cure in connection with displacements I mean that the parts remain in their normal position when the pessary has been removed.

(2) Retroversion of the uterus occurring in young and nulliparous women, when the symptoms of displacement are of recent origin, and the uterus fails to keep up after simple reposition. In such cases I think it is always advisable to try the effect of a Hodge pessary in the first instance, and give it a year's trial if necessary.

(3) Retroversion of the gravid uterus in the early months; this is the class of case in which the patient often seeks relief on account of impaction of the retroverted uterus and retention of urine. It is usually sufficient for the pessary to be worn for six or eight weeks. This is the only kind of retroversion in which I prefer a ring to a Hodge; the primary necessity is to keep the uterus raised up, for if raised it will not fall backwards. In cases of retroversion of the unimpregnated uterus I have hardly ever known a ring to correct the displacement.

In all cases of backward displacement it is an indispensable condition that the diplacement should be corrected before the pessary is introduced.

(4) Prolapse of the uterus, with or without cystocele, when the perinæum is sufficiently good to hold a ring well up. If the ring is well borne, and does not readily get rough or set up vaginitis, and if it relieves the symptoms, it may be worn for a considerable time with periodical inspection, particularly if the patient is single or a widow.

(5) Cystocele with prolapse, with the same proviso as above, that the perinæum is sound. Sometimes a ring answers very well; in other cases a specially shaped pessary may be adopted, the upper end of which is like a Hodge, while the lower end is widened and curved forwards so as to lie behind the pubes and above the levatores ani.

(6) All kinds of mobile diplacement when the condition of the patient is such as to contra-indicate operation. In this category I should include some cases of displacement in old women, although, if the patient's general condition is vigorous, I should not consider age in itself to be a bar. I have had some very satisfactory cases of operative treatment for displacement in women over seventy.

Whilst I am on the subject of pessaries I should like to call your attention to a few popular delusions:

That the vagina was designed by Nature as a receptacle for a pessary.

That the introduction of a pessary is good treatment as a placebo when one does not know what else to do.

That a Hodge pessary may be expected to succeed when the retroversion has not been first rectified with the fingers or sound.

That a ring is a suitable pessary for a backward displacement.

That the size and fit of a pessary are unimportant.

I now pass on to my second group.

(*b*) Cases in which there is a Clear Indication for Operation.

In this category I should include the following:

(1) All cases of backward displacement in which the uterus is fixed by adhesions, so that its position cannot be rectified. To insert a pessary in such a case is merely to add risk to inefficiency.

(2) Backward displacement complicated by ovarian tumours or inflammatory disease of the appendages. It may be said that in such a case the ovarian tumour or tubal disease, and not the displacement, is the indication for the operation. With this I quite agree, but the points upon which I would lay stress are that it is futile in such a case to treat the displacement by pessaries; and that when the abdomen is open one should use the opportunity to sling up the uterus if the operative conditions allow of this being done.

(3) Backward displacement complicated by one or more fibroids admitting of myomectomy. Here the fibroids may, in certain circumstances, be considered as the indication for operation; but there are also cases in which the fibroids might not be regarded, in themselves, as calling for immediate interference, and I should then regard the double condition as a clear indication for a combined myomectomy and hysteropexy.

(4) Mobile retroversion associated with torn perinæum that requires repair. While the patient is under the anæsthetic, it is a simple matter to open the abdomen and deal with the uterine displacement; it does not add materially to the operative risk; it takes very little longer; and the patient is saved from the necessity of subsequent pessary treatment.

(5) Retroversion associated with an atrophic condition of the upper part of the vagina in such a fashion as to render pessaries ineffectual.

(6) Prolapse of the uterus and vaginal walls, when the perinæum is so torn or stretched that no ordinary pessary will keep in place.

(7) Total procidentia, with the vagina inverted and the uterus lying outside the vulva; particularly when associated with ulceration of the vagina due to friction of the clothes. In such cases the vaginal outlet is nearly always so stretched that pessaries fail to keep in place; and moreover, if a pessary can be induced to stay in, it will nearly always cause excoriation and ulceration of the damaged vaginal wall.

(8) All cases of displacement when the patient is going to reside abroad in districts where medical assistance is not readily available.

(9) All cases of displacement in which a pessary rapidly perishes or becomes rough, or leads to profuse and offensive discharges, or causes inflammation or ulceration of the vaginal walls. I have met with such cases, when the patient was absolutely miserable without a pessary, and yet had to do without it at frequent intervals because it was so badly borne. There has then been an alternation of (1) relative comfort, but associated with offensive discharge; and (2) freedom from discharge but with attendant discomfort and inability to retain urine when walking, coughing, or straining in any fashion.

(10) Cases of displacement in neurotic single women, on whom the concentration of attention on the pelvic organs incidental to the wearing of a pessary, frequent douchings and periodical examinations has a harmful mental and moral effect.

(*c*) Optional Cases, in which the Patient should have Both Courses explained to Her, and be allowed to Choose.

I place in this group cases of uncomplicated displacements in which a pessary answers quite well as regards keeping the displacement corrected and the patient comfortable, but does not bring about a cure. As soon as the pessary is removed the displacement returns. Consequently there is before the patient the prospect of many years of pessary treatment. Now *à priori* one would expect, and from experience of many such cases I can testify, that to many women such a prospect is abhorrent. They do not want to be constantly reminded of the existence of their internal organs; the periodic examinations are distasteful to them; the constant douching necessary to keep a pessary clean is nothing short of a nuisance; and they accept with surprising alacrity the suggestion that they may undergo an operation to rid them of this great incubus in their life. In this class we find married women of some years' standing; young married women to whom a pessary appears as a barrier between them and their husbands and their hopes of happy maternity; and single women to whom every examination is a veritable ordeal.

I maintain that in the past the requirements of these patients have not been adequately met; they

have had to put up with an objectionable treatment, or "grin and bear" their pains and discomforts unrelieved. If we can open to them a door of relief and, instead, bar it in their face, we fail to do our duty. There is a perfectly fair parallel in the case of hernia in men. If a man with a hernia came to us and said that he did not wish to wear a truss but preferred to undergo a radical cure, even though he realised the slight risk attendant on this procedure, we should without hesitation put him in the way of obtaining his wish. The wearing of a truss by a man is a far slighter hardship than the wearing of a pessary by a woman.

The only valid argument against operative treatment of displacements as a matter of election would be one that could demonstrate that the operative risk was excessive, or that the results were bad. The risk in these days is very slight: I have published my results fully from time to time. In my first ten years I had 3 deaths in 64 cases—all from broncho-pneumonia. In the last ten years I have had 3 deaths in 509 cases—one from diabetes, one from oral sepsis and one from pneumonia. I submit that these figures would compare not unfavourably with any series of 500 cases of radical cure of hernia. As regards the later results of operation, I have reported from time to time the after-histories of my own cases. The latest report was in the discussion at the Royal Society of Medicine, in March of this year, when the total cases traced amounted to 373—a larger number than those reported so far in the practice of any other operator. The figures are large enough to make the deductions from them trustworthy; they showed—

(1) That in 90 per cent. of cases the general condition of the patient was much better than before operation; (2) that in 96 per cent. of all the cases, and in 98 per cent. of the later ones, the displacement was permanently cured; (3) that 62 confinements had followed the operation; with two more natural confinements that I have recently heard of, the number is now 64, in none of which has there been any complication that could be attributed to the operation; (4) that the uterus had maintained its good position in spite of pregnancy and labour in 97 per cent. of cases; (5) that the patients had been free of bladder disturbance, or had no more than before operation in 85 per cent. of cases, 20 per cent. having actually improved.

From the facts given it is clear that any argument against the operative treatment of displacements, based on the assumption that the operative mortality was high or the after-results bad, would fall to the ground.

One speaker in the discussion referred to expressed the opinion that these operations were being done much too frequently. Such a position is as unsound as religious intolerance. The cure of the patient is the one aim of medicine and surgery; and if one man finds that he gets the best results from one method, and another finds he is most successful using another method, neither has the right to say of the other that he is not justified in using his method, or to impose any limitations as to how often he may use it.

The correct attitude is to keep an open mind: to adopt such methods as appear to be giving the best results; and to abandon those methods whose results are inferior. Success in treatment is the final arbiter in all therapeutic measures; and I am convinced by experience that in the treatment of uterine displacements they will meet with the greatest success who recognise that pessaries, while they have their place, have also their limitations; and are prepared to avail themselves of the resources of surgery in suitable cases.

December 22nd, 1913.

Erythema Nodosum and Tuberculosis.—V. Lovati records two cases of erythema nodosum in children, aged 7 and 10 respectively. The former showed signs of tuberculosis of the left apex, and in the latter there was no definite tuberculous focus, though the general condition was suggestive of latent infection. Both gave a positive cuti-reaction. While admitting the existence of some cases of idiopathic erythema nodosum, Lovati holds that it is the manifestation of a soil suited for tuberculous infection, which, if not actually present, may develop at a more or less distant date. —*Brit. Journ. Child. Dis.*, vol. x, p. 549.

THE TREATMENT OF PHTHISIS AND HÆMOPTYSIS BY THE INDUCTION OF AN ARTIFICIAL PNEUMOTHORAX.*

By CLAUDE LILLINGSTON, M.B., B.C.

THE origin of the treatment of phthisis by a pneumothorax is due to the observation that progress of tuberculous disease in a lung is occasionally arrested by the development of an accidental pneumothorax. Usually this occurrence is fatal in a few hours or days, for the pleural cavity often becomes infected with pyogenic organisms through the lesion in the visceral pleura, and this lesion is often valvular, permitting the entry of air into the pleural cavity without affording it an exit. Air is, therefore, sucked into the pleural cavity without being able to escape. Hence the distressing dyspnœa so often accompanying an accidental pneumothorax. When an accidental pneumothorax is beneficial, it may be assumed (1) that no germs, other than tubercle bacilli, have reached the pleural cavity, and (2) that the pneumothorax is large enough to compress the diseased area of the lung effectively. An accidental pneumothorax is sometimes so limited by adhesions that it scarcely causes any discomfort. I have lately seen a patient of Dr. Burton Fanning's whose cough one day was followed by subcutaneous emphysema, which appeared over the second intercostal space, and spread thence to the shoulders and arms. Evidently air had escaped through both layers of the pleura, which must have been adherent over the lesion. These adhesions may have saved the patient's life at the time, but they also prevented the induction of an artificial pneumothorax at a later date.

The credit for first suggesting the conversion of a clinical disaster into a therapeutic success is due to the Englishmen Carson and Caley in the last century; but it is to Forlanini of Pavia, Murphy of Chicago, and Saugman of Denmark that the credit is due for the evolution of the treatment from its primitive stages to its present position. Now it has gained such a position that it is practised in every important continental and American sanatorium and hospital for diseases of the lungs, and is the subject of a large output of medical literature. The bibliography on this subject now includes about 500 "original" contributions, and a society formed at the International Medical Congress this summer has undertaken to publish, in three languages, a journal devoted exclusively to the abstracting and reviewing of all the papers published on this subject throughout the world.

The scope of this paper is far too small for every aspect of this treatment to be mentioned, far less discussed. I will confine myself to three points—the rationale of the treatment, the choice of patient, and the results of the treatment.

RATIONALE.

The improvement which a pneumothorax effects is primarily due to the immobilisation of the inflamed part, the diseased lung being treated on the same principles as a tuberculous knee. In both cases movement, and therefore auto-inoculation, are reduced to a minimum by the enforced rest imposed by splints. Another beneficial factor is the expression and drainage of fluid from the lung, which, when completely collapsed, is scarcely larger than a human kidney. Dr. Lewthwaite attributes the frequent loss of weight observed during the first few weeks of the treatment, in part at any rate, to the expression of fluid from the collapsed lung. Even when adhesions prevent complete collapse, a partial pneumothorax may greatly facilitate the drainage of cavities, and the expression of fluid from the lung. Another favourable influence is the permeation of the tuberculous foci in the lung with scar-tissue, which appears to encapsule diseased areas more rapidly than when spontaneous cicatrisation leads to a clinical recovery. Fortunately cicatrisation is practically limited to tuberculous areas of the lung; and healthy areas have been found to retain their capacity for complete re-expansion after a pneumothorax has existed for several years. Other and more fanciful explanations have been put forward to explain the beneficial action of a pneumothorax, but they seem somewhat fantastic and far-fetched.

* Read at the East Anglian Branch of the British Medical Association.

Choice of Patient.

Only about 5 to 10 per cent. of all phthisical patients are suited for the treatment. This sounds disappointing, but these patients are recruited from otherwise hopeless material, and the results are often startlingly good. The condition which calls most urgently for the treatment is extensive and active disease on one side, with slight, quiescent disease on the other. The ideal is unilateral disease, and I have often been unable to detect any sign of disease on one side; but the tell-tale evidence of the dead-house reduces the frequency of true, unilateral disease, in the presence of active disease on one side, to a negligible quantity. The most suitable case is that of a young adult who has suddenly collapsed with a smart hæmorrhage or signs of pneumonia. In a few weeks the whole of one lung is involved; dulness, crepitation, and diminution of breath-sounds are evident over the whole lung, the temperature is 100° to 102° F. every evening, the pulse is about 100, the expectoration is profuse and teems with tubercle bacilli, the cough is troublesome, night-sweats are severe, and the patient is cyanosed and dyspnœic. The other lung is apparently healthy, and the larynx and abdomen are not involved. Let us assume that this condition has persisted for a couple of months, in spite of rest in bed, at a properly conducted sanatorium. The prognosis in such a case under any treatment but that of an artificial pneumothorax is bad. But provided the diseased lung is not adherent to the chest-wall, a pneumothorax will reduce the temperature to normal in a week, and in nine cases out of ten the patient will be leading an active life a year later.

Unfortunately such a case is rare, and it is more common to find patients with bilateral disease, but with far more advanced and active disease on one side than on the other. Let us take the case of a man whose illness has lasted a couple of years, during which he has been unable to work, and his temperature has been alternately febrile, subfebrile and normal. Sanatorium treatment has been carried out all the time, but the cough and expectoration have persisted, and the disease has slowly progressed. We will suppose that the disease began in the left apex, and extended to the third rib in front, and to the spine of the scapula behind. Later, this cleared up partially, while the disease spread throughout the upper two thirds of the right lung. At the end of the second year of illness there is dulness over the apex of the left lung, respiratory sounds are harsh, expiration is prolonged, and an occasional post-tussic click is audible. Over the rest of the left lung there is rather loud compensatory breathing, suggesting that this lung is already doing some of the work of its fellow. Over the right lung there is dulness, with bubbling *râles* audible everywhere except at the base, where respiratory sounds are very faint. The excursions of this side of the chest are less than those of the opposite side. The prognosis is less easily made in this than in the former case. If the patient can afford to leave his work for an indefinite period, he may ultimately regain partial capacity for work; but it is more probable that he will die in a year or two. As the left lung has already taken over much of the right lung's work, the additional work thrown on it by the collapse of the right lung is not likely to upset it. Indeed, if the formation of toxins in the right lung can be arrested by its compression, the chances for the left lung are improved, even though it is given hard labour. If, therefore, there are no pleural adhesions, a right-sided pneumothorax will probably arrest the disease on both sides.

Sometimes it is advisable to induce a pneumothorax in the absence of fever if the patient's disease is still progressing, or stationary but rendering him unfit for work. Fever is, however, the best index to the action of the pneumothorax, and patients submit more readily to its discomforts when fever, which has lasted for months and has been refractory to various forms of treatment, falls within a few days of the first injection of gas, and remains normal thenceforth.

Let us now suppose another case in which the history is much the same as in the above case, but the examination shows signs of quiescent disease in the left lung extending from the apex to the fourth rib in front, and to the angle of the scapula behind. The right lung shows signs of cavitation and breaking-down from the apex to the same level as on the left side. The question raised will be: Can the left lung do the work of two without breaking down again? In such a case the opinion of the physician who has attended the patient for many months is invaluable, for he will have observed the behaviour of the left lung, and have formed a shrewd opinion of its condition, whether

there is real or only apparent arrest of the disease. It is often impossible to anticipate the results of a pneumothorax in such a case, and if the treatment is attempted it is advisable to point out its tentative character to the patient to avoid subsequent disappointments. My experience in such cases has been rather baffling; patients with signs of fairly extensive though more or less latent disease in the sounder of the two lungs have gained a new lease of life by the treatment; while in two cases I have seen the disease flare up in the sounder lung, although scarcely a sign of disease was demonstrable in this lung before the pneumothorax was induced. Probably extensive but quiescent disease in the sounder lung is a much less serious obstacle to a pneumothorax than a small focus of active disease; and if the sounder lung has recently shown progress of the disease, it is advisable to defer the induction of a pneumothorax till it is again quiescent on this side.

Recently I was asked to induce a pneumothorax in a middle-aged man whose right lung had broken down two years earlier. The disease had slowly become quiescent on this side, when, in the spring of this year, he developed pleurisy on the left side, which had previously been almost well. When I saw him in August he was still febrile, but less so than in the spring. He had scarcely any expectoration, his appetite and general vitality were good, and he was not emaciated. His right lung appeared to be a mass of fibroid tissue and cavities, except at the base, where normal respiration was audible. The left lung showed no sign of the pleurisy and was apparently healthy, except at the apex, where harsh respiration and crepitation were audible in front and behind. To collapse the right lung would mean overworking the left lung, in which the disease, though slight, was probably active; and to collapse the left lung would mean throwing additional work on a lung the greater part of which was a fibroid mass. In either case, a pneumothorax would almost certainly have hastened the course of the disease, which, with care and time, may become permanently arrested without an operation.

So far, only cases of advanced disease have been discussed. Is a pneumothorax to be recommended in the earlier stages of the disease? I think not, as a rule, for pulmonary tuberculosis is usually curable in the early stages by real, not pseudo-sanatorium treatment. But there are a few exceptions. The first is violent or persistent hæmoptysis, which either threatens the patient's life or his mental stability. There are many patients whom "Bluidy Jack," as Stevenson termed it, has reduced to a state of neurasthenic misery. Last winter a patient of Dr. Leonard Ley's at Yarmouth was subject to recurrent attacks of hæmoptysis which rendered him unfit for work. The disease was limited to the right apex, where there were signs of a cavity. But below the third rib in front and the spine of the scapula behind the lung seemed healthy. The left lung also seemed healthy. The hæmoptysis ceased after the first injection of nitrogen, and the patient, who has resumed his work, is still having his pneumothorax maintained by Dr. Ley.

Another class of patient who is suitable for the treatment in the first stage of the disease is not seldom seen in sanatoria. There are signs of slight dulness over one apex, with crepitations which come and go, and never extend below the second rib in front or the spine of the scapula behind. The other lung is apparently healthy. In spite of a year's treatment in a sanatorium the physical signs have not changed, tuberculin has done only harm, the temperature has been subfebrile for several hours after slight exercise, the appetite has been poor, there has been no gain of weight, and the patient is flabby and on the high road to hypochondria and valetudinarianism. In such a case a pneumothorax may be a Godsend.

In another case the disease may involve only the apex of one lung and the larynx. The cough is troublesome and gives the larynx no rest. By the arrest of the pulmonary disease by a pneumothorax the cough may cease as if by magic, and the laryngeal disease show rapid improvement. A patient sent by Dr. Burton Fanning to Mundesley Sanatorium illustrates this point admirably. The whole of his left lung was involved and both vocal cords were ulcerated. Dr. Pearson, of Mundesley Sanatorium, tells me he has not hitherto seen such a bad case of laryngeal disease terminate in recovery. But as soon as the diseased lung was collapsed, the cough and temperature dwindled, and the laryngeal condition improved steadily. Two years later, the vocal cords are practically healthy, the patient is afebrile and leading a fairly active life. Of course, laryngeal disease is not an indication

per se for the induction of a pneumothorax, but so far from being a contra-indication, as was at one time held, its existence, combined with pulmonary disease, sometimes hastens the decision for an artificial pneumothorax. Tuberculous disease of the intestine, on the other hand, when associated with persistent diarrhœa, is certainly a contra-indication of the treatment. So also is amyloid disease.

Results.

I have treated with an artificial pneumothorax twenty-six patients, and have failed to induce a pneumothorax in eight other patients on account of pleural adhesions. In most of these cases I have started the pneumothorax at various sanatoria, and the injections have been continued by the resident medical officers. I have not, therefore, kept in touch with most of the patients. My material is accordingly unsatisfactory for analytical purposes, and it is also too small considering what a capricious disease pulmonary tuberculosis is. Far more satisfactory material for analysis is given in Prof. Saugman's paper on one hundred cases read before the International Congress in London this year. He limited his observations to the patients in whom the treatment was prescribed more than nineteen months earlier. An effective pneumothorax was induced in sixty-four patients; in thirty-six pleural adhesions prevented the formation of an effective pneumothorax.

As the patients in these two classes differed only in having or not having extensive pleural adhesions, a comparison of their subsequent fate is a useful index to the value of the treatment.

	Pneumothorax effective.	Pneumothorax prevented by adhesions.
Able to work	32	8
Unable to work	18	12
Dead	14	15
Unknown	0	1
	64	36

A percentage analysis of these figures shows that more than twice as many patients regain their capacity for work in the first as in the second class, and that the cases terminate fatally twice as often in the second as in the first class. In the first class tubercle bacilli disappeared in 50 per cent., in the second class in 8·6 per cent. Striking though these figures are, they yet do not impress the mind so strongly as one typical case observed before and after treatment.

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December 22nd, 1913.

Fatal Peritonitis following the Use of a Laminaria Tent.—Hussy reports a case, from von Herff's clinic, aged 45, who had begun to be irregular in her menses. This, however, was not imputed to the menopause but to a combination of myomatous uterus with impending abortion. After ergot and a tamponade had accomplished nothing the house-physician introduced a laminaria tent. Soon a chill, followed by fever, set in. The face was pale and anxious. Hypogastrium tender. Uterus unevenly enlarged. Diagnosis, myomatous uterus, beginning peritonitis. There was a suspicion that the swelling tent had caused perforation. Exploratory laparotomy revealed purulent peritonitis. The uterus and adnexa were removed, but patient soon died from generalised peritonitis. The initial tamponade no doubt paved the way, and as a result of dilatation with the tent germs ascended into the tubes and peritoneal cavity. —*Med. Record*, vol. lxxxiii, p. 1000.

THE TECHNIQUE OF OPERATIONS FOR INTESTINAL OBSTRUCTION IN THE LARGE BOWEL, WITH ILLUSTRATIVE CASES.

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FOR many years the results of operations for intestinal obstruction due to growths in the large intestine were a reproach to surgery. Little progress was made until the importance of first draining the distended intestines was realised before attempting the radical part of the treatment. As the text-books give scanty details on the subject, an account of the routine practice now in vogue at the Royal Victoria Infirmary, Newcastle-on-Tyne, may prove useful to those surgeons who have not yet adopted this method, and instructive to students as the correct course to follow in these cases.

When the causes of the high mortality hitherto found in statistics of intestinal obstruction due to growths in the large bowel were sought for, two facts stood out prominently:

(1) Too often the case was allowed to go on unrelieved, and only when the patient was almost beyond the aid of surgery was the unfortunate subject sent into hospital.

(2) Too much was attempted by the surgeon at the operation.

Let us for a moment briefly consider the history and appearance on examination of these cases.

History.—The patient has noticed that for some time there had been a difficulty with the bowels alternating with diarrhœa, the difficulty becoming increasingly more marked and accompanied by "rumblings." Steady loss of weight, marked impairment of appetite culminating in an attack of subacute or acute obstruction, with vomiting coming on late in the case.

Examination of the abdomen reveals marked and uniform distension, visible peristalsis, and when the peritoneal cavity is opened the whole length of the intestine down to the seat of obstruction is hypertrophied, distended, congested, and in the worst possible condition for resection and subsequent anastomosis.

Under the old *régime* the abdomen was opened in the middle line and the cause of the obstruction dealt with then and there. Before this could be done great difficulty was experienced owing to the mechanical encumbrance offered by the distended intestine, and far too much manipulation took place, leading to paresis of the bowel.

Often the loaded coils had to be eviscerated, then emptied of their fluid contents before it was possible to find the obstructing cause. Under such circumstances it is easy to understand that radical attempts to remove the cause of obstruction were not likely to succeed, and the introduction of the septic element by opening the intestine still further prejudiced the chances of success.

The contents of the bowel were virulent and the lines of suture more likely to become infected and yield, with consequent diffusion into the peritoneal cavity.

Even in cases not markedly obstructed from mechanical causes, the contents of the bowel in the neighbourhood of a foul, malignant ulcer are so virulent that it is difficult to guard against local or general sepsis unless preliminary drainage is employed. *Obviously the safer and more rational method of treatment is:*

(1) To drain the distended intestine above the site of obstruction by a preliminary cæcostomy.

(2) Allow the patient to recover from his auto-infection.

(3) Explore at a second operation; find and remove the cause if possible, restoring of the intestinal channel.

(4) Closure of the cæcostomy opening.

Drainage of the intestine.—This is best brought about by performing cæcostomy, tying in a Paul's tube and allowing the intestine to empty itself. The reason for choosing cæcostomy is that in a large proportion of cases the growth is found in the neighbourhood of the sigmoid section of the colon. If the colon was opened in the left iliac region, this area would be fouled for the second stage of the operation, and a favourable termination not so likely to ensue.

It may be asked, does not fæcal matter accumulate below the opening in the cæcum and convert the bowel into a cesspool? A certain amount of fæcal matter seems "to cake" on the wall of the

bowel below the cæcostomy opening and above the obstruction, but the effects of this can easily be counteracted when the distension has disappeared by washing the interior of the gut out daily. To accomplish this a tube of soft india-rubber is passed along into the colon through the cæcostomy opening, and through this tube a stream of warm boracic lotion or saline solution is directed. Another rubber tube may also be inserted *per rectum* and the stream of lotion directed through the stricture. In the course of two or three days the bowel interior is cleansed.

The time allowed for the patient to recover from his auto-infection.—This varies according to the condition of the affected person, but usually in two to three weeks the tongue has cleaned, appetite has returned, and the intestine has regained tone. There is no hard and fast rule, the general condition of the patient being the best guide.

The patient is now in a fit state to submit to the second stage—viz. *exploration for the cause of obstruction, resection of the growth and restoration of the continuity of the alimentary canal.*

The incision.—Either a median incision below the umbilicus or, if the growth can be palpated, in the sigmoid—an oblique incision over the left iliac fossa, downwards and inwards.

The median incision is preferable when no growth can be felt, because it is easier to explore the whole length of the colon through this opening, and the presence of secondary nodules or wide dissemination of the disease can be readily ascertained.

Resection and anastomosis.—If the growth is removable the bowel is clamped as far from the seat of disease as possible by two pairs of intestinal clamps, above and below the two incisions necessary for removal.

It is necessary to keep in mind that if possible sufficient bowel should be left to restore the continuity of the alimentary canal.

The bowel is best divided by means of the actual cautery at a dull red heat, for by this means the edges of the divided gut can easily be sterilised before closing them. If the cautery is not handy or does not work properly, division with a knife and sterilisation of the cut edges by pure carbolic acid is a good substitute.

The ends of the divided gut are closed, either by tying a silk ligature round, and then burying this by means of a purse-string suture through the peritoneal and muscular coats, and re-inforcing this by a few interrupted Lembert sutures, or by a continuous catgut suture in two lines, one near the margin of the divided gut, the other at a deeper level, with inversion of the end as before.

Restoration of the continuity of the intestinal canal.—If there is not enough spare bowel it may be necessary to do an end-to-end union, but lateral anastomosis is unquestionably the safer method when the local conditions allow of its performance.

Sometimes it is found impossible to obtain enough bowel for either end-to-end or lateral anastomosis; then a permanent left inguinal colostomy with closure of the cæcostomy opening is the proper course to follow.

The wound is now closed, and if the anastomosis is satisfactory and free from tension the cæcostomy opening may be closed at this stage. Less strain is thrown on the site of anastomosis by not closing the cæcostomy opening until all danger of yielding is passed.

Closure of the cæcostomy opening may be undertaken with safety after ten days from the restoration of the alimentary canal.

What is known as "mobilising the colon" will often convert a doubtful into a safe anastomosis. This is effected by dividing the peritoneum on the outer side of the large bowel—a place devoid of blood-vessels of any importance. The division is made parallel with the bowel. There is a vessel in the fold which holds the splenic flexure, and this may require a ligature.

Many urge against this method the formidable prospect of two or three operations and the disinclination of the patient to face it. The best answer to this objection is that statistics amply prove that it is safest and attended with a much lower mortality. Having said this, no further defence is necessary.

The surgeon must judge for himself the propriety of shortening the three operations to two, but it is well to remember that the treatment carried out in three stages is the safest course.

In the following list of cases this technique has not been slavishly adhered to because of the conditions found, but as a routine practice no surgeon will regret employing it.

CASE 1.—*Acute Intestinal Obstruction due to Carcinoma of the Pelvic Colon; Exploratory Laparotomy and Cæcostomy; Resection of the Growth; Lateral Anastomosis; Recovery.*

A female, æt. 40 years, was admitted into the Royal Victoria Infirmary under my care on April 25th, 1913, suffering from acute intestinal obstruction.

On examination patient was very ill, the abdomen distended; nothing could be felt *per rectum*.

First operation: April 25th, 1913.—A median laparotomy was performed; a growth palpated in the sigmoid; the intestines were distended. The median incision was closed and a cæcostomy was done with insertion of a Paul's tube.

Second Operation: May 14th, 1913. — The median wound was re-opened; a growth of the pelvic colon was found adherent to the left Fallopian tube, ovary and broad ligament. The growth, together with the tube and ovary, was resected, the ends of the gut closed, and a lateral anastomosis performed.

The cæcostomy wound was left untouched.

Result.—The patient picked up very slowly, and on June 18th she had recovered sufficiently to go home. It was not deemed prudent to do any further operation to close the cæcostomy opening. She returned on August 11th in splendid health with the cæcostomy opening healed over, though there was slight bulging.

Microscopic report. — Typical columnar-celled carcinoma.

CASE 2.—*Acute Intestinal Obstruction due to a Carcinoma in the Pelvic Colon; Cæcostomy; Exploration; Resection of Growth and Tying Tube into Proximal End; Closure of Cæcostomy Opening; Lateral Anastomosis and Closing Colostomy Wound; Recovery.*

A female, æt. 35 years, was admitted under my care into the Royal Victoria Infirmary on July 21st, 1911, suffering from acute intestinal obstruction.

History.—She had suffered from mild attacks of colicky abdominal pain. Since January the pains became more severe and frequent and progressive constipation followed.

Two days before admission she was seized with severe pain, increasing in intensity, accompanied by borborygmi and absolute constipation.

On examination there was marked abdominal distension and visible peristalsis *per rectum*; nothing felt.

First operation: July 21st, 1911.—Cæcostomy done and Paul's tube inserted.

Second operation: July 23rd, 1911.—Abdomen opened in middle line; a growth of the pelvic colon found; patient collapsed, so was hurriedly closed.

Third operation: August 10th, 1911.—Growth excised; patient became collapsed, and a Paul's tube was inserted into the proximal end of the resected gut.

Fourth operation: August 25th, 1911. — Cæcostomy wound closed.

Fifth operation: Lateral anastomosis and closure of the colostomy opening.

Result.—Recovery.

Microscopic report of growth.—Carcinoma.

CASE 3.—*Carcinoma of the Pelvic Colon; Excision of the Growth and Cæcostomy; Lateral Anastomosis; Closure of Cæcostomy; Recovery.*

The Mother Superior of a Dominican convent, æt. 47 years, was sent to a gynæcologist, who asked me to attend the operation as he thought the case was one of a growth in the bowel.

History.—For the last year she had suffered from constipation, which has got steadily worse, and accompanied by paroxysmal pains, and latterly she has vomited. There has been a steady loss of weight. A doctor in the south felt a tumour in the pelvis a year ago.

Examination under ether revealed the presence of a nodular mass separate from the uterus and movable. There was slight distension.

Diagnosis. — Malignant bowel or possibly an ovarian tumour.

First operation: November 16th, 1912.—The abdomen was opened in the middle line below the umbilicus and a growth of the pelvic colon found. There was slight distension of the small and large intestine and no evidence of dissemination. The growth was excised, the ends closed, and as the patient was not in good condition and slight distension was present no anastomosis was done; the median wound was closed. Cæcostomy was performed through an incision in the right iliac region.

Result.—The median wound healed *per primam*, and after two weeks the large bowel was washed

out daily through the cæcostomy opening and also *per rectum*. Much stagnant fæcal matter was evacuated.

The specimen was a "ring-carcinoma."

Second operation: December 15th, 1912.—The abdomen was re-opened in the middle line; the sigmoid was mobilised by dividing the peritoneum on the outer side; a lateral anastomosis was performed with difficulty owing to the sparsity of the lower end of the bowel. The median wound was closed and the cæcostomy left untouched.

Result.—The operation was successful; there was a little superficial suppuration.

Third operation: January 11th, 1913.—The cæcostomy wound was closed and healed by first intention.

January 31st.—The patient went back to the convent feeling well, with the bowels acting normally.

CASE 4.—*Acute Intestinal Obstruction due to a Carcinoma in the Pelvic Colon; Cæcostomy; Exploration, Left Inguinal Colostomy, and Closure of the Cæcostomy; Recovery.*

A male, æt. 43 years, came into private hospital on April 23rd, 1913, suffering from acute intestinal obstruction.

History.—For two years he has not felt well and was much troubled with flatulence.

September, 1912.—He commenced to suffer from "diarrhœa," at first twice a day, then gradually increasing in frequency. His body-weight commenced to diminish.

April 22nd, 1913.—Acute intestinal obstruction came on with vomiting and absolute constipation.

On examination.—He was thin and vomiting everything. The abdomen was markedly distended.

Per rectum.—Nothing could be felt.

First operation: April 23rd, 1913.—A cæcostomy was done and a Paul's tube inserted.

Second operation: May 6th, 1913.—The abdomen was opened in the middle line; an irremovable mass was found in the pelvic colon firmly fixed to the brim of the pelvis and causing complete obstruction. The median wound was closed, left inguinal colostomy performed and the cæcostomy wound closed.

Result.—The median wound healed *per primam*; there was a slight leak in the cæcostomy wound. He went home greatly improved.

CASE 5.—*Intestinal Obstruction due to Carcinoma of the Splenic Flexure of Colon, Cæcostomy and Exploration; Lateral Anastomosis between Transverse and Pelvic Colon; Closure of Cæcostomy Opening; Recovery.*

A female, æt. 46 years, was admitted under my care into the Royal Victoria Infirmary, August 12th, 1913.

History.—She was quite well up till three months ago. She then had an attack of severe pain in the abdomen and similar ones have continued intermittently since. The attacks of pain have been accompanied by vomiting and the pain is general across the abdomen. She has lost a little flesh and strength.

First operation: August 13th, 1913. — The abdomen was opened through the right rectus muscle and the transverse colon was distended. A fixed growth was felt at the splenic flexure, but on account of the distension the abdomen was closed, cæcostomy performed in the right iliac region and a Paul's tube inserted.

Second operation: August 26th, 1913.—Median laparotomy performed with excision of the umbilicus; a large fixed carcinoma found at splenic flexure, not deemed removable. Lateral anastomosis was then done by simple suture between the transverse and pelvic colon.

Third operation: September 13th, 1913.—The cæcostomy opening was closed.

Result.—Patient recovered.

December 22nd, 1913.

FIBROIDS OF THE UTERUS: THEIR PATHOLOGY, DIAGNOSIS AND TREATMENT. By Sir JOHN BLAND-SUTTON, F.R.C.S. Science Reviews Limited. Pp. 245, with 39 Illustrations. Price 4*s.* 6*d.* net.

"In this little book," to quote from the preface, "an attempt is made to set down in narrative form a summary of our knowledge of the extremely common tumours known as uterine fibroids." Like everything else from the pen of the author, this book is very readable and contains a great deal of information about these tumours, but the large section of degenerations is almost completely ignored.

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SOME POINTS IN THE TREATMENT OF GRAVES'S DISEASE.*

By FREDERICK LANGMEAD, M.D., F.R.C.P.,
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OUR knowledge of the causes of exophthalmic goitre is not yet so advanced that we are able to treat the disease with scientific precision. The indications which an understanding of its ætiology would provide are to so large an extent wanting that much of the treatment which is employed is directed against the consequences rather than against the original cause of the malady. Although there are a few notable exceptions, the great majority of writers on this subject now regard the symptoms of Graves's disease as due to hyperthyroidism arising from increased activity of the thyroid gland. Treatment may therefore logically be considered under four heads:

(1) Medical measures, which may be employed to diminish the activity of the thyroid gland.

(2) Those devoted to counteracting the excessive thyroid secretion.

(3) Those employed to lessen or abolish the consequences of that secretion (symptomatic).

(4) Surgical procedures.

(5) Treatment directed against the underlying cause.

(1) MEASURES TO DIMINISH THE ACTIVITY OF THE THYROID.

The first of these, namely, *rest*, is one which is universally employed. The good effects which so commonly follow complete rest in bed can, I think, be explained—at least in part—by the decreased metabolism which necessarily follows,

* A lecture delivered at the Polyclinic.

and in which the thyroid takes part. Another factor which may play a part is the diminished activity of the circulation when complete rest is enjoined, which would tend not only to lessen the amount of secretion which the thyroid gland pours out, but also the rate at which it gets into the circulation. By lessening metabolism also, it is possible that it may lessen the work of the thyroid as an antitoxic gland—a question to which I intend to refer later.

A *diet* largely consisting of milk is usually found to be the most suitable, and possibly acts by lessening thyroid activity. Proteins should be diminished for the same reasons, and, moreover, Dr. Chalmers Watson's experiments show that they influence the structure of the thyroid gland. During exacerbations of thyroid activity when the characteristic symptoms of Graves's disease become rapidly more severe, the application of *cold*, as by an ice-bag directly to the thyroid gland, also brings about a considerable but temporary improvement. *Certain drugs*, which have been proved to be of distinct value in the treatment of the disease, such as arsenic and belladonna, possibly have a chemio-therapeutic effect and act directly upon the thyroid gland. In many hands *X rays* have been of decided advantage. Dr. Florence Stoney (1), in particular, has published very favourable results from their use. They need, however, very careful application, as dermatitis is easily produced.

Dr. George Murray (2), of Manchester, describes a case which suggests the way by which X rays may act. In a case under his care eleven doses of X rays were given between November 28th, 1911, and January 5th, 1912. On January 9th the right lobe of the gland was removed because the patient had a severe attack of dyspnœa with loss of consciousness. Microscopical examination of the portion removed showed a diffuse inter-alveolar fibrosis, which, in places at any rate, appeared to be of recent origin and to be the result of the action of the X rays. At the time of the operation the capsule of the gland was found to be unusually adherent to the trachea and surrounding parts, which Dr. Murray also ascribed to this form of treatment. If this inference is a true one, it is conceivable that the good effects which follow their use in some cases which have received protracted treatment are due to fibrosis.

Electrical treatment by faradic current applied directly over the gland for two or three hours daily is also occasionally beneficial and may also act by lessening its activity.

A more novel attempt to cripple the excessive activity of the thyroid gland has been made by Dr. Beebe (3), of New York. He showed by experiment that if a tissue nucleo-proteid—for example, that of liver or kidney—were injected into an animal, its serum contained a cytolysin which was highly destructive to the tissue injected. He therefore injected into rabbits the nucleo-proteid of the thyroid and the thyreo-globulin. Having obtained the serum, he has used it in the treatment of 2000 cases of hyperthyroidism, and has certainly obtained some very good results. But from a review of his cases it is clear that many could not properly be considered as examples of Graves's disease. Moreover, sera actively cytolitic are known to possess a common disability, in that they may cause destruction of cells other than those for which they are intended.

(2) Measures Directed to Counteracting the Excess of Thyroid Secretion.

Attempts have been made to neutralise the excess of thyroid secretion by the administration of the *sera, dried blood or dried milk* of goats and sheep from which the thyroids have been previously removed. The last substance, desiccated milk, sold by the name of "rodagen," has been fairly extensively used. The principle which has led to their employment is as follows: It has been supposed that the excess of thyroid secretion is due to an effort of the thyroid gland to neutralise poisons circulating in the blood, an effort which has more than accomplished its aim and led to the presence in the blood of more thyroid secretion than is required. The blood and milk of the thyroidectomised animal has been thought to contain these toxins in excess, and they have been given to patients suffering from Graves's disease to neutralise their excess of thyroid secretion. Little success has attended their use. The reputed antagonism of the suprarenal gland has also formed a basis of treatment along these lines. The late Dr. G. A. Gibson, of Edinburgh, in his address in medicine to the British Medical Association in 1912, said that in his opinion no remedy approached, even distantly, *suprarenal extract* in importance, and that it might be regarded as almost specific

for this affection. Other observers have not obtained such good results, and, according to Weiner, administration of adrenalin causes an increase of thyroid secretion.

(3) Symptomatic.

Drugs are, of course, frequently found useful in controlling some of the symptoms of the disease. Thus in cases where great nervous excitability leads to loss of sleep, associated with much palpitation, *opium* is often of considerable value. It is contra-indicated, however, when the disease has advanced sufficiently to have produced degeneration of the myocardium. Another drug which is commonly used to control tachycardia and cardiac palpitation is *digitalis*, but it is open to question whether this drug does not do more harm than good by increasing the strength of the muscular contraction in an organ already over-stimulated. *Bromides* in certain cases certainly quieten the patient's excitement, but their action can only be considered as temporary and produces no continuous improvement. *Atropine* may be employed to lessen the discomfort of those in whom sweating is a prominent symptom. A serious symptom which calls for more careful treatment than it usually receives is glycosuria. It should be treated as carefully as glycosuria of other forms, for if allowed to continue the clinical picture of diabetes in its graver form may be produced, and may ultimately lead to a fatal issue.

(4) Surgical Treatment.

A great deal has been written in recent years concerning the surgical treatment of Graves's disease, and it has formed the subject of many important discussions, such as one which took place in London for the Royal Society of Medicine last year.

To estimate the value of operative procedures we must first get a clear idea of the results which attend medical treatment alone. There is no doubt that the impression which we derive from text-books as to the mortality of the disease is altogether too gloomy. Dr. Hector Mackenzie (4), speaking from a large experience, is probably nearer the truth. He considers that roughly about 25 per cent. of the cases make a complete recovery, 25 per cent. recover, but bear some signs of their former illness, about 25 per cent. live on as chronic invalids, and 25 per cent. die from the disease.

Dr. Hale White (5) has gone carefully into this question. He investigated the after-histories of 102 hospital and private cases, treated between 1888 and 1909, and submitted his figures to Mr. Thiselton, who gave him an actuarial report. Of the hospital patients, the "expected deaths" among women of the age implicated during that time were 5; the actual deaths among the patients were 8. Taking only those between the ages of 30 and 45, and excluding the few cases before and after those ages, "expected deaths" came out at 3 and actual deaths at 5. Including the private cases also, it appears that 15 actually died, whilst 8 could be counted as "expected deaths," so that the mortality of patients with exopthalmic goitre was apparently twice as great as that of people in general. As Dr. Hale White points out, it must be remembered also that all these patients were sufficiently ill either to obtain admission to hospital or to seek a second opinion. On the other hand 11 had been operated upon, and 4 died as the result. Only one reported herself as feeling perfectly well; the others were much better, but not cured. The cases selected for operation were usually mild.

There can be no doubt that the operative mortality of this disease is high. Mr. James Berry (6) collected 97 cases for which surgical measures had been employed in recent years in the various London hospitals, and among them the deaths numbered 16, 12 of which occurred within 48 hours after the operation.

The operative mortality of those who have had a very extensive experience, such as Kocher of Berne and the Mayos of Rochester (Mass.), is considerably less than this, but I learn from one who has had an opportunity of seeing the work of these surgeons, that many of the cases which appear in their statistics are not such as we are accustomed to label as frank examples of Graves's disease.

Wilfred Trotter (7), from a study of the Continental literature on this subject, tells us that the fatality of the first 50 cases operated upon by Continental surgeons is usually about 10 per cent. As the result of improved technique, the greater facility acquired by practice, and a careful selection of cases, this death-rate diminishes in each surgeon's practice, and Trotter believes that it may be expected to be diminished by half, and reach a percentage of 5.

With these figures as to the relative mortality of

cases treated solely by medical means, and those subjected to surgical operation before us, I think we can say with justice that surgery cannot be accredited with reducing the number of deaths from this disease.

The justification for the employment of surgery must, I think, be based, not upon the mortality results, but upon the effect which it may have in relieving the patient's symptoms and shortening the duration of the illness. The lot of sufferers from this malady is often a very hard one; the character of their symptoms is such as to give little peace of body or of mind, and after protracted illness some 25 per cent. recover only to such an extent as to remain chronic invalids for the rest of their lives. During the active stage such patients, despite their nervousness and anxiety, are willing to submit to any measure which promises relief, and surgery, dealing as it does in no uncertain manner with the source of the hypersecretion, is often able, I am convinced, to fulfil such a promise.

I have had an opportunity during the last six months or so of seeing several cases which Mr. Berry has operated upon at the Royal Free Hospital, both before and after the operation, and although at first somewhat biassed against the surgical treatment of this affection, I am bound to confess that it brings amelioration of symptoms, often in a most convincing manner, and affords a valuable means of staying the progress of the disease.

Profoundly modifying the good effects or otherwise of this treatment is a most careful selection of cases and of the time for operating. There is, I think, no circumstance under which it is more advisable that physician and surgeon should work hand-in-hand. Those who have had the greatest experience and the best results are the most careful in the selection of their cases, and the most chary of operating in the presence of certain contra-indications. Then Kocher (8) holds that special attention should be paid to the heart, kidneys, liver, adrenals, pancreas, lymphatic organs (thymus, spleen, lymph-glands), and to the blood. A constantly irregular pulse, a lasting notable amount of albuminuria, very frequent fatty stools, distinct and constant glycosuria, *status lymphaticus* (especially if unaccompanied by the usual lymphocytosis), a very great increase of lymphocytes in the blood combined with marked leucopenia and very slow coagulation of the blood are in his opinion factors which forbid an operation unless they can be made to disappear under proper treatment during the preparatory stage preceding operation.

The Mayos of Rochester work in close conjunction with Dr. Plummer, who examines all the cases of Graves's disease in consultation. According to Mr. Berry (9), Dr. Plummer considers it important to gauge the degree and stage of the thyroid intoxication and to avoid operation while it is at its height. He holds that the majority of the cases run a fairly definite course. "If the average course of the disease be represented by a curve, the greatest height of the intoxication is found to be reached during the latter half of the first year and then rapidly drops to the twelfth month. In many instances it reaches the normal base line during the next six months; more often it fluctuates with periods of exacerbation for the next two to four years. Secondary symptoms and exophthalmos may remain, but the active course rarely continues over four years without distinct intermissions. The ascent may be gradual, sudden, or irregularly marked with secondary curves." Dr. Plummer advocates operation either quite early in the disease or much later, when the period of greatest intoxication is over. At Rochester the average duration of the disease at the time of operating is about two years. Some cases are rejected altogether. Mr. Berry (10) has told us that in estimating the degree of intoxication both the Mayos and Plummer lay great stress on the amount of mental excitability and muscular weakness which are present. The latter is often marked in the quadriceps, and is tested by asking the patient to step up on to a low stool. They regard dilatation of the heart to the extent of one inch as a serious matter, and dilatation to 1½ in. as necessarily associated with a percentage of unavoidable mortality.

Mr. Berry (11) considers that operation should not be undertaken in acute cases in which there is much thyroid intoxication, as shown by great excitability, mania or muscular weakness, or in which any acute inflammatory affection such as bronchitis is present. Secondly, when definite degenerative changes have taken place in the disease, especially the heart and kidneys. He advises postponement of the operation in the

presence of albuminuria, glycosuria, diarrhœa, a constantly irregular pulse, and low blood-pressure. If these symptoms persist in spite of medical treatment, operation, in his opinion, should not be undertaken. "Of all chronic complications," he says, "marked dilatation of the heart is perhaps the most common and the most serious."

I have dealt at some length with the practice of those surgeons who have had a considerable experience of the operative treatment of the disease, in order to emphasise the great degree of caution which they use in the selection of their cases. If we consider the many reservations which they make, it is clear that in their opinion, at least, there are many cases where the risk is too great for this form of treatment to be employed. At a time when doctors are beginning to recommend patients affected with Graves's disease to have an operation performed, no matter what the stage of the disease, and are making somewhat extravagant promises as to the expected result, I have thought it well to put these facts clearly before you. Cure seldom follows. Relief is often obtainable, but careful medical examination and a great degree of caution are necessary to make the result worth the patient's risk.

(5) Treatment of the Underlying Cause.

All the methods of treatment which are recounted above, whether directed to lessening the activity of the thyroid, to neutralising the hypersecretion, to relieving the symptoms which accompany it—or indeed, surgical treatment—are, after all, merely symptomatic. The underlying cause of the undue thyroidal activity is not considered. Until we understand more fully what produces the hypersecretion, we must often rest content with what is, after all, subsidiary therapy, namely, the treatment of its effects.

For the solution of this problem, the greatest hope at present seems to be connected with investigations into the antitoxic properties of the thyroid gland. Rupert Farrant's (12) work strongly suggests that the hyper-activity is an evidence of its reaction in an attempt to destroy circulating toxins. Thus he found (1) that the thyroid undergoes hyperplasia in certain diseases, and that a similar hyperplasia is produced in guinea-pigs by the injection of diphtheria toxin. This is mitigated if the guinea-pigs have thyroid administered to them combined with the diphtheria toxin and the animals survive longer than the controls. (2) The blood-serum of a thyroid-fed rabbit is antitoxic to diphtheria toxin. (3) Antitoxin fed to normal rabbits produces symptoms similar to those arising from feeding them with thyroid, whilst in thyroid ectomised rabbits antitoxin is borne without symptoms. (4) Diphtheria antitoxin contains iodine in organic combination; normal horse-serum contains only the slightest trace. This, he points out, indicates some close relationship between the thyroid function and the development of certain antitoxins. "It may be suggested that the hyperplasia observed in these toxæmias arises from an attempt to form antitoxin."

It is interesting that the condition found in the thyroid when its reaction to toxins was at its acme is similar to that which occurs in Graves's disease.

This work and the history of cases certainly strongly suggest the possibility that the hypersecretion of the thyroid is in the nature of a reaction to circulating toxins. At any rate they give an indication strong enough to be acted upon, and show the importance of the recognition of Graves's disease at its very beginning. This hypothesis holds out hope that at the earliest stage treatment devoted to sources of intoxication, whether from the teeth, tonsils, bowel or elsewhere, may prevent the development of the disease into its classical clinical form.

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(2) 'Lancet,' February 24th, 1912, p. 489.
(3) 'Interstate Med. Journ.,' St. Louis, 1912, vol. xix, pp. 409–414.
(4) 'Lancet,' March 2nd, 1912, p. 576.
(5) *Ibid.*, February 24th, 1912, p. 504.
(6) *Ibid.*, March 16th, 1912, p. 725.
(7) *Ibid.*, March 9th, 1912, p. 641.
(8) *Ibid.*, March 2nd, 1912, pp. 576–579.
(9) *Ibid.*, March 8th, 1913, p. 670.
(10) *Ibid.*
(11) *Ibid.*
(12) 'Proc. Roy. Soc. Med.,' 1912, vol. vi (Path. Sect.), pp. 21–48.

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RENAL INFANTILISM.*

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Of recent years a good deal of attention has been paid to the subject of infantilism, or asexual ateleiosis. By these terms is meant a condition of retarded development in which the growth of the sexual organs is particularly backward, the latter characteristic being the point of differentiation of this class from sexual ateleiosis, in which the dwarf is of the "small adult" type. This delay in development has been traced to numerous causes, the imperfect functioning of various organs or the presence of different diseases. By these means subgroups of infantilism have been described. From the consideration of recent cases reported there can be recognised a type of infantilism associated with, and apparently due to, a perversion of the renal functions. These cases are alike only in that they appear to be due to the perversion of the renal functions, for the evidence shows that the actual condition of the kidneys is not the same in all cases. The title of "renal infantilism" has the advantages of being sufficiently broad to include all the cases, and of bringing the nomenclature of this class into line with that of other known types of symptomatic infantilism which are dependent on the abnormal functioning of other organs, *e.g.* intestinal, pancreatic, hepatic infantilism.

Symptomatology.

The degree of infantilism present is variable. In most of the cases where organic renal disease is present it has been marked, the child being of a stature corresponding to a considerably younger age. Thus in one case, the patient, æt. 9¾ years, was only a trifle taller that his normal brother of 3½ years. In another the patient of 9 years was shorter than his brother æt. 5 years. The mental development usually corresponds to the stature rather than to the age of the patient. Where no organic disease is present in the kidneys the infantilism is of a less severe grade. Genu valgum has been noticed in several of the cases. It may be regarded as evidence of the imperfect osseous development which obtains in other types of infantilism.

Of more interest are the special characteristics of the renal class of infantilism. Of these the most prominent are polydipsia and polyuria. The thirst is severe, and may be the cause of the patient's coming under observation. The polyuria is marked, sometimes extreme, as in a case in which the patient passed in twenty-four hours a weight of urine equal to one fourth of his body-weight Bed-wetting, repeated two or three times nightly, is likely to be present.

As the result of the polyuria the skin of the patient is very dry and the face rather characteristically wrinkled. This is shown in Fig. 1, where even the professional photographer has failed to obliterate all the lines of the face. The complexion is often of a pale yellow tint. In some cases marked anæmia is present.

In the group of cases without organic renal changes (diabetes insipidus) all the symptoms are of a less severe type than where chronic renal disease is present.

The age at which the symptoms were first noticed by the parents is of considerable interest In many cases the polydipsia and polyuria were stated to have existed since birth, and in these growth was impaired from infancy onwards. In others the child was stated to be normal for the first few years of life, the thirst, polyuria and retarded growth manifesting themselves at some period of childhood.

The urine is much increased in amount, a daily output of 50–70 oz. being common. It is of very low specific gravity (1002–5). The other changes in the urine depend upon the actual condition of the kidneys and will be described later.

Cardio-vascular changes are present in some cases and are of much interest (*vide* ætiology of Group 1). They consist of hypertrophy of the left ventricle of the heart, heightened blood-pressure and thickening of the arterial walls, which may be first recognisable in the brachial arteries.

* A revised edition of an article in the 'British Journal of Children's Diseases' (1912, vol. ix, p. 289). Now published by request, with permission of the Editor of that Journal.

Classification.

On a pathological basis cases of renal infantilism may be divided into two groups:

Group 1: Renal infantilism with organic renal disease (chronic interstitial nephritis).

Group 2: Renal infantilism without organic renal disease (diabetes insipidus).

These two groups will now be considered separately.

Group 1: Renal Infantilism with Organic Renal Disease (Chronic Interstitial Nephritis).

In the cases to be described in this group there will be seen to be many similarities and only two points of difference. These latter consist of the presence or absence of cardio-vascular changes, and the age at which the symptoms were first noticed. These clinical differences are of considerable interest, which is enhanced by the fact that they do not appear to correspond to any pathological differences. Autopsies, as will be shown in the section dealing with the morbid anatomy of this group, have been made in the main types of case falling under this heading, and in each one the same changes—those of chronic interstitial nephritis—have been found

(a) *Symptoms Congenital; no Cardio-vascular Changes recognisable.*

Of this type of case, the most interesting subdivision of Group 1, we have two examples. The first quoted is that recorded by Dr. H. Morley Fletcher, to whom belongs the credit of being the first to point out this association of symptoms. Case 2 is of extreme interest in that it tallies in almost every detail with Case 1, and coming to autopsy, was found to be chronic interstitial nephritis.

Case 1 (Fletcher [1]).—Boy, æt. 6 years; has not grown since the end of his first year. Polyuria since birth; at four months is said to have taken four pints of fluid daily. Genu valgum developed at five years.

Family history.—Eldest child; one sister, healthy. Mother has phthisis. Has had one miscarriage.

Present state.—Height, 2 ft. 7 in.; weight, 21 lb. Wearing clothes made for him when two years old. Drinks about 3½ pints daily. Intelligence good. Testicles undescended. Urine: amount, 40–70 oz. daily; sp. gr. 1005; albumen, 0·16 per cent.; granular casts occasionally present. Kidneys palpable. Cardio-vascular system normal; blood-pressure, 75 mm. Hg. Inherited syphilis, no clinical evidence; Wassermann negative.

Course.—Thyroid extract given without improvement.

Case 2 (Miller).—Girl, æt. 6 years.

Past history.—Polyuria noticed since birth; later would wet the bed two or three times in the night, soaking through more than one mattress. Thirst not noted until child was weaned at eight months, when she required an abnormal amount of fluid. At no time in her life did she pass a night without waking up in order to drink. At birth she was extremely small—said not to be more than 2 lb. as a guess. She was not short-coated until she was eight months old, and she was nicknamed "Dolly" on account of her small size. At three years she was admitted into St. Mary's Hospital with diarrhœa and vomiting. She was then noted to be very small, weighing only 14 lb. Heart normal. Urine not recorded. Did not walk until she was four years old; no knock-knee. She was backward mentally, saying only short words.

Family history.—Youngest child. One brother, æt. 10 years, healthy, showing no signs of syphilis. Between son and patient there were two miscarriages, at four and ten weeks respectively. Mother says she was very weak from the latter miscarriage when she became pregnant with patient. Mother: Wassermann reaction, negative.

Present state.—Patient only 3 ft. high. Admitted to hospital with uræmic convulsions. No hypertrophy of the heart could be recognised then or when seen two days previously in out-patient department, where she had been brought up for five days' constipation, and was treated with castor oil. Complexion very sallow. Body small, thin, and yellow in colour. Child died two hours after admission. No urinary examination made.

Autopsy.—Chronic interstitial nephritis found; very slight hypertrophy of left ventricle (*vide* "Morbid Anatomy").

Although in Case 2 no examination of the urine was made, there is no reason to doubt, considering the clinical history and the condition of the kidneys, that it would have corresponded exactly to the urine in the other cases of this group.

(b) *Symptoms Congenital; Slight Cardio-Vascular Changes recognisable.*

The next case is of interest in that it seems to form a connecting link between the above cases in which no cardio-vascular changes could be recognised, and the next type in which such changes are marked. In it the left ventricle was slightly but definitely enlarged, the blood-pressure raised to 130 mm. Hg., and the brachial arteries were unduly palpable. A further point of interest in this case lies in the fact that the child was nearly ten years old, the oldest of those reported in whom the symptoms have been noted since birth.

CASE 3 (Miller [3]).—Boy, æt. 9¾ years. Full-term child, said to be a good size at birth. Polyuria and polydipsia since birth; worse since an attack of diarrhœa at five months. Grew very little; still on bottle at three years; not put into breeches until four years old on account of small size. Measles at four years. Knock-knee developed when he began to walk.

Family history.—Parents' ages at birth of child—father thirty-three, mother thirty. Fifth in family of six, others normal. No deaths, no miscarriages.

Present state.—Height 3 ft. 1½ in.; is only a trifle taller than his brother æt. 3½ years (*vide* Fig. 2). Proportions according to Stratz's tables are those of a child of under six years. Weight 34 lb. Intelligent but very childish, the mental development being in keeping with his size rather than his age; speaks well, can understand well, can read very little, can write his own name. Is still in infants' school. He is markedly anæmic; his face is sallow and wrinkled (Fig. 1); lips very pale.

Genito-urinary system.—Kidneys not enlarged; marked phimosis. Testes descended, small. Urine: Passes 40–60 oz. in hospital daily; sp. gr. 1001–4; persistent trace of albumen, only once absent, lessened slightly by rest in bed; by Esbach, 0·02 per cent.; urea 0·65 per cent. On one occasion there was a very large number of granular casts, on others few or none. Urine sterile.

Cardio-vascular system.—Slight enlargement and hypertrophy of left ventricle, clinically and by skiagram; apex-beat just external to nipple-line. Pulse-rate persistently 100–120. Radial arteries not thickened, but brachial quite palpable. Blood-pressure by mercurial manometer 130 mm. Wassermann reaction negative.

FIG. 1.—Case 3 (on right), aged 5½ years, with normal brother, aged 8 months; showing wrinkled skin and sunken eyes.

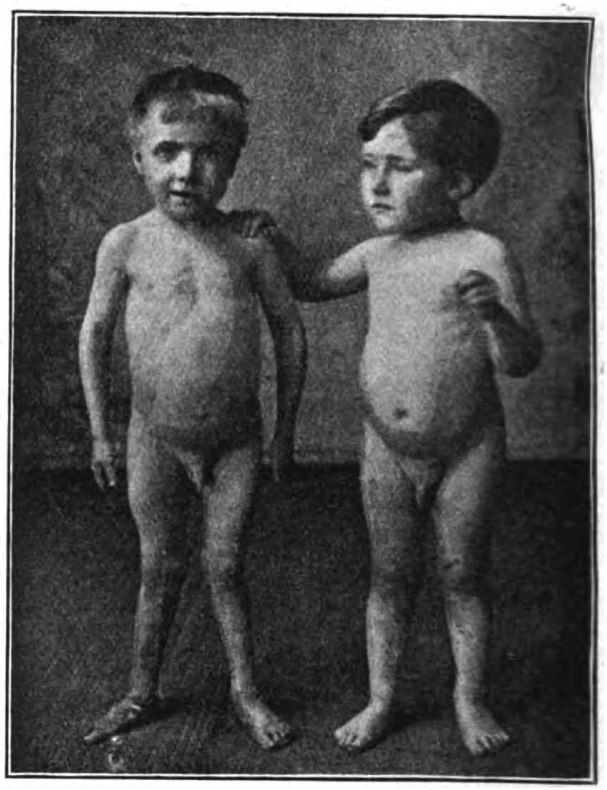

FIG. 2.—Case 3 (on left), aged 9¾ years, with normal brother, aged 3 years; showing short stature and knock-knee.

Course.—Under occasional observation for two and a half years; very little change. Grew about

an inch in stature. Symptoms less marked, probably owing to education. Pallor increased. Thyroid extract given without benefit. Child died (March, 1912) of acute pneumonia; no autopsy made.

(c) *Symptoms Congenital; Marked Cardio-vascular Changes Recognisable.*

The next two cases show well-marked cardio-vascular changes at the ages of six and a half and three and a half years; in both the symptoms existed since birth.

CASE 4 (Parsons [5]).—Girl, æt. 6½ years. Small at birth and during infancy. Polyuria and polydipsia since birth.

Present state.—Physically and mentally a child of three years. Heart hypertrophied; œdema present. Broncho-pneumonia developed and rapidly proved fatal. Urine (during terminal illness): Sp. gr. 1010; faint trace of albumen; no casts.

Autopsy.—Broncho-pneumonia; chronic interstitial nephritis; hypertrophied left ventricle (*vide* "Morbid Anatomy").

CASE 5 (Parsons).—Girl, æt. 3½ years. Polyuria and polydipsia since birth; grew little, sat up late; weight at three years, 16 lb.

Family history.—Fifth in family of six; others normal.

Present state.—Physical and mental development of child of eighteen to twenty-four months. Weight 20 lb. Pale, cyanosed, wasted. No genu valgum. No evidence of syphilis. Heart shows some hypertrophy of left ventricle. Arteries normal. Œdema variable, sometimes of face, feet, legs, vulva with ascites. Urine: amount varies with œdema; sp. gr. 1001–4; faint haze of albumen, occasionally absent; no casts; urea 0·8 per cent.

(d) *Symptoms originating after Birth.*

This type of case does not appear so common as the foregoing as a cause of infantilism. In the three cases of this type, the first, reported by Dr. Otto May (2), appears to have been normal for the first four or five years, after which, with the onset of polyuria and polydipsia, development became much retarded, and the general condition of the patient simulated that of those described above. The other two (Cases 7 and 8) have been recorded by Dr. Naish (4).

CASE 6 (May [2]).—Boy, æt. 9 years. Normal at first, but has hardly grown since he was between five and six years old. Never robust. Suffers from polydipsia, polyuria and enuresis.

Family history.—Fifth in family of seven.

Present state.—Corresponds to a child of four to five years; brother of five years is taller and heavier than patient. Complexion: yellowish skin, very dry and wrinkled. Urine: passes 700–1600 c.c. daily (average 1250 c.c.); considerable cloud of albumen; occasionally a few granular casts. Heart normal; brachial arteries more palpable than normal; blood-pressure 60–100 mm. Hg. Wassermann reaction negative.

CASES 7 and 8 (Naish [4]).—Boys, æt. 16½ and 9½ years. They appeared about ten and four years old respectively. Both had genu valgum developing at fourteen and eight years old. Both were small at birth and very much smaller than the other members of their families, who were all apparently normal. Both had "polyuria and polydipsia for a considerable portion of their lives."

Autopsies.—In both there was a condition of chronic interstitial nephritis present (*vide* "Morbid Anatomy").

This type of case, therefore, strongly resembles the types in which the symptoms are congenital in every way except the age at which the symptoms develop. So close is the resemblance, indeed, that it may be that in these cases the early symptoms have escaped the parents' notice, and that in reality the symptoms were present from birth. Certainly, however, we cannot conclude that this is so, although such a view is rather tempting. There is, on the other hand, no theoretical difficulty in accepting the possibility of post-natal cases similar in every other way to congenital cases.

MORBID ANATOMY OF GROUP 1.

Five out of the eight cases recorded here have died, and of these, four (Cases 2, 4, 7 and 8) have been examined pathologically. In these, full clinical and pathological data are available for comparison. It is noteworthy that in Case 2, cardiac hypertrophy was not recognisable during life and was barely certain after death, whereas in the others it was definite; yet, contrary to expectation, the renal changes in all the cases were almost identical. Case 2 is of further interest in that it died of uræmic convulsions.

The morbid anatomy in Case 2 will be described first and the changes in the other cases compared to it.

Case 2.—(Symptoms congenital; cardio-vascular changes not recognisable during life; death at six years from uræmic convulsions.)

Thymus degenerated. Brain congested. Lungs congested. Liver slightly fatty. Spleen congested. Adrenals, pancreas, alimentary tract, genitals and glands normal.

Heart.—Pericardium normal. The cavity of the left ventricle was not enlarged, but comparing the wall of the left ventricle with that of another child of six years, it was thought to be very slightly hypertrophied. Myocardium appeared healthy, firm and of good colour. Endocardium normal. Arteries normal.

Kidneys.—Both very small, weighing together just over 1 oz. Length 4·3 and 4·75 cm., breadth 3 cm. They both presented an exactly similar appearance. The capsules were scarcely at all thickened, and stripped very easily, leaving a coarse granular surface. There were no cysts, the granules being solid and consisting apparently of compensatory hypertrophy. Stellate veins injected. On section the parenchyma was firm and pale. The cortex was greatly diminished in width (4–6 mm.), though there was some compensatory hypertrophy between the pyramids. The pyramids were firm and fibrotic. Organs anæmic but vessels prominent. Pelves slightly granular. Ureters normal. Bladder contracted, appeared normal.

Histology of kidneys (*vide* Fig. 3).—The chief change is in the interstitial tissue, which is much increased in amount. The new-formed tissue is highly cellular, and large accumulations of small round cells are scattered throughout the section. The vessels are moderately thickened, though the vascular changes are not extreme. The glomeruli are much altered and vary in size and shape. A few are completely sclerosed, and may show fibrotic changes with adhesions of the tuft to the

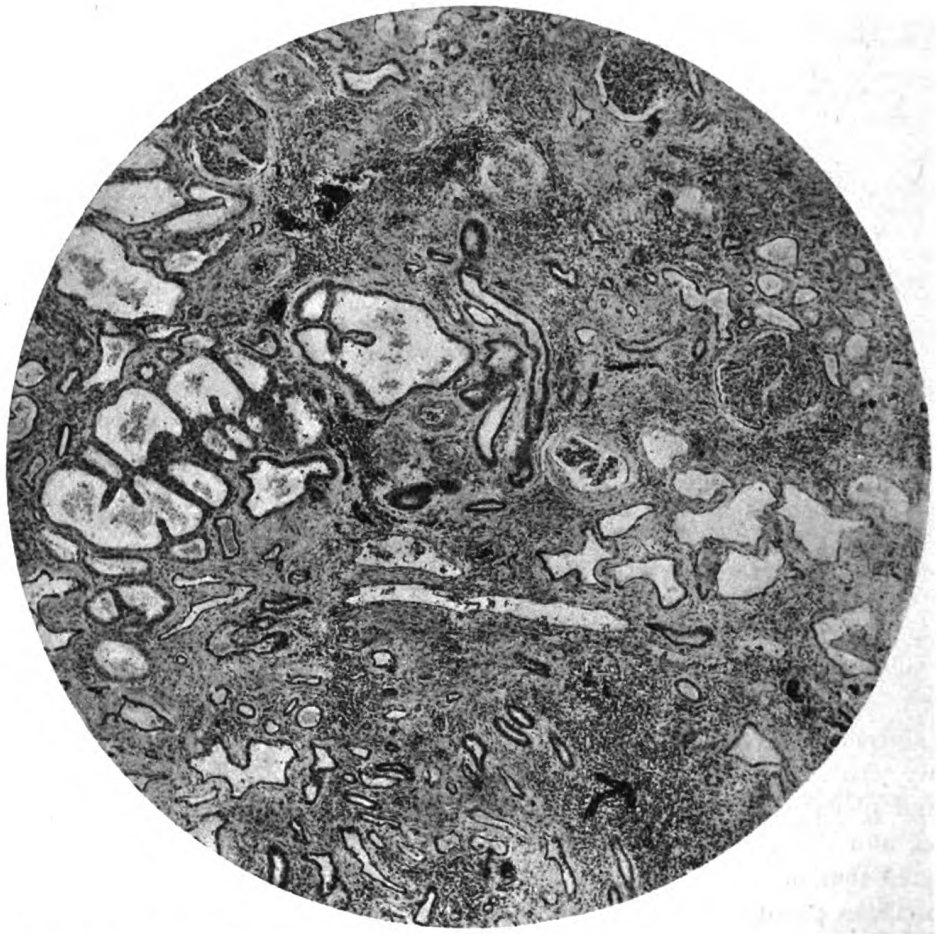

Fig. 3.—Microphotograph of section of kidney (Case 2), showing active interstitial nephritis, etc. (× 50.)

capsule. Some show a little hyaline change. The condition of the tubules appears to be secondary to the interstitial change. They are irregularly dilated, some are almost cystic, but the epithelium is healthy except for some flattening due to pressure. A few of the convoluted tubules contain cell *débris*, and in some of the collecting tubules there are masses of red blood-cells, but in the main there appear to be no active parenchymatous changes.

(For the above description we are indebted to Dr. E. H. Kettle, Assistant Pathologist, St. Mary's Hospital.)

Case 4 (5).—(Symptoms congenital; marked cardio-vascular changes; death at 6½ years from broncho-pneumonia.)

Heart.—Cavity of left ventricle enlarged; wall markedly thickened (¼–⅜ in.). Microscopically myocardium showed some excess of fibrous tissue.

Kidneys.—In appearance similar to those in Case 2; weight of both together just under 1 oz. Microscopically the changes in the parenchyma, glomeruli and arteries were identical with those in Case 2; the interstitial changes appeared of a slightly earlier stage than in Case 2, but were otherwise quite similar.

Cases 7 and 8 (4). The histology of the kidneys in one case, D—, was very similar to Case 2; in the other, W—, they were also very similar but rather less marked.

Ætiology of Group 1.

Of the eight cases recorded, five were males and three females. In five the symptoms were present from birth, and in three they were said to develop during early childhood. In this connection it is interesting to recall that Dr. J. E. H. Sawyer found that the sex-incidence in the granular kidney of childhood was in the proportion of two males to three females. Out of his forty-six cases three were said to have had symptoms since birth (6).

The type of organic renal disease causing this group of cases of infantilism is, it seems clearly established from the evidence we have procured, that of chronic interstitial nephritis, the kidneys in all the cases that have been examined pathologically being very closely similar. That there should be no differences in the renal lesions in the congenital and post-natal cases presents no particular difficulty to our minds; we do not know the cause of the nephritis, and can well suppose that in one type it is the result of some toxæmia acting during intra-uterine life, and in the other the result of a toxæmia acting at a later period of life. But that there should be no pathological differences in the renal conditions whether the hypertrophy of the heart be marked or so slight as to be barely certain even after death, is, we think, not a little surprising. Yet a study of Cases 2 and 4, which are very strictly comparable in every particular except the condition of the heart, proves that such a difference does not necessarily denote a different form, or severer degree, of renal inflammation. In Case 2, the interstitial nephritis was the more marked of the two and the child actually died of uræmic convulsions, and yet the hypertrophy of the heart was infinitesimal. The condition of the general nutrition of the children does not appear to us to account for this difference, and we confess that we have no explanation to submit.

Such nephritis is in children commonly regarded as being most frequently due to inherited syphilis. In the cases here brought forward there is no evidence of the presence of this disease; in many, indeed, such a possibility can be entirely excluded. To us it seems impossible that inherited syphilis should produce such a marked disease of the kidneys without showing signs in the other organs, at least when examined post mortem. Thus out of the eight cases, three that were not examined pathologically gave a negative Wassermann reaction, while in two others at least (probably four) there were no signs of syphilis post mortem. On these grounds, therefore, we can reasonably hold that renal infantilism with chronic interstitial nephritis is not necessarily due to inherited syphliis.

Lastly, as regards the cause of the retarded development in these cases, it has been suggested that this is to be explained by the diminution or absence of an internal renal secretion. While this may be so it seems unnecessary to us to invoke such a theory, and we would be content to regard the stunting of the growth as due to excessive drainage from the persistent polyuria and albuminuria.

Group 2: Renal Infantilism without Organic Renal Disease (Diabetes Insipidus).

In this type of renal infantilism the interference in the physical and mental growth appears of less severity than in the group associated with chronic

interstitial nephritis, and the association between infantilism and diabetes insipidus has received very little attention.

Diabetes insipidus is an uncommon condition, particularly in children. Futcher (8), reporting a series of nine cases, notes only one in a young subject, a girl of thirteen years.

The following case, which has been under the occasional observation of Dr. G. A. Sutherland at Paddington Green Children's Hospital during the past ten years, and which is here reported by his kind permission, shows some interesting points in connection with diabetes insipidus, the congenital onset of the symptoms, the occurrence of several cases in two generations.

Case 1.—Female, æt. 16 years.

Past history (obtained partly from step-mother, who has had charge of patient since her third year). —Abnormal thirst ever since birth. Size at birth not known; certainly small for her age at three years, but could walk and run well then. Was very backward at school; never did well at her lessons.

At six years (1902) was admitted to Paddington Green Children's Hospital under Dr. Sutherland for thirst and wasting. Was noted to be very undersized as well as very thin. She seemed to have an unlimited capacity for taking fluid, though was not distressed if it were withheld. The amount of urine passed varied closely with the amount of fluid taken; on 80 oz. in a day she passed 77 oz., on 46 oz. she passed 37 oz., etc. The urine was of a very light colour, specific gravity about 1004, and always free of albumen and sugar. Under treatment she got a little fatter. She was stated to be bright and happy in the ward.

At ten years of age she was readmitted for the same complaints, and showed no change from the condition reported above.

Family history.—Mother is stated to have had "drinking diabetes" all her life; died at forty-five; had thirteen children, of whom only five lived, and several miscarriages. Two of patient's brothers have had "drinking diabetes" all their lives; one, now aged twenty-seven, is only 5 ft. high, is married, and has one child; the other, aged twenty-three, is tall. Another brother, unaffected, died of pulmonary tuberculosis.

Present state.—Patient was traced and examined (1912). Is now sixteen years old, and suffers from thirst "just the same as before." She will drink a couple of pints during most nights, and will easily drink a quart in a few moments. She will still drink dirty water as she used to do when a child. Has had no incontinence for past three years. She is said to have grown rather more rapidly recently, and is now 4 ft. 11 in. high. She still looks very childish and is dull and listless, although answering questions correctly; memory appears good. Step-mother says she is like a child of ten mentally; she is said to be very stupid at work. Circumference of head 20¼ in. Breasts are rather small for her age; axillary and pubic hair is present, but menstruation has not yet started.

Heart appears normal, but radial arteries are distinctly thickened; blood-pressure 108 mm. Hg. Lungs normal. Thyroid normal in size. No signs of organic nervous disease.

Urine said to be much increased in amount; sp. gr. 1001; no albumen, sugar or casts. Wassermann-reaction negative.

The next case, one recently reported by Dr. F. Parkes Weber, shows many points of similarity with the above, but no symptoms were noticed until the third year of life.

Case 2 (Weber [10]).—Boy, æt. 10 years. Polyuria and abnormal thirst since third year. Incontinence to seventh year. Eldest of family of eight; others unaffected; one stillbirth immediately following patient.

Present state.—Small for age; height 3 ft. 9½ in. (normal 4 ft. 3¾ in.). Weight two thirds of normal. Thyroid small. Cardio-vascular system normal. Skiagram of skull shows sella turcica of normal, or possibly increased, dimensions. Urine: passes 4000 c.c. in twenty-four hours; sp. gr. 1001–4: no albumen or casts. Wassermann reaction positive. No improvement under treatment with thyroid, mercury, or salt-free diet.

The next case illustrates the connection between diabetes insipidus and hydrocephalus, examples of which are well known. The growth of the patient seems to have been much interfered with, which perhaps warrants its mention here. Such a complicated case illustrates what we should imagine to be the fact, that renal infantilism with diabetes insipidus does not form so clear an entity as the type with chronic interstitial nephritis. Evidently some of the cases might be classed under the disease originating the symptoms (syphilis, pituitary

lesions, gross organic nervous lesions) rather than as renal. Possibly inherited syphilis would account for the symptoms in this case.

Case 3 (Cherry [7]).—Female, æt. 2½ years. At the age of two child suffered from diarrhœa, vomiting and intense thirst. The former symptoms lasted one week only, but the thirst has persisted. She will drink one and a half gallons daily. Loss of weight was noted. Three months later the child fell on to back of head, after which enlargement of the head and exophthalmos developed. One month later deafness supervened. The abdomen became much enlarged and the liver hypertrophied. Weight of patient at two and a half years 22 lb. (normal 33 lb.).

The only reference to diabetes insipidus as a cause of infantilism which we can trace in the literature of the subject, with the exception of that by Dr. F. Parkes Weber already referred to (10), is an article by Pechkranc published in 1911 in Warsaw, entitled "Diabetes Insipidus; also Imperfect Development of the Entire Body and of the Genitals" (9). We much regret that we have not been able to procure this.

Ætiology and Symptoms of Group 2.

We do not propose to enter into the question of the ætiology of diabetes insipidus. The disease is well known to be connected in many cases with pituitary and other gross lesions of the nervous system, with syphilis, acquired or inherited, while some cases appear to be entirely "functional" in origin. We should suppose that diabetes insipidus, arising from any cause, if it developed early enough and was sufficiently severe and protracted, could retard the physical and mental development of the patient.

The infantilism with diabetes insipidus is not of so severe a grade as that found associated with chronic interstitial nephritis; the stunting of the growth is not so obvious, nor is the dried, wrinkled appearance so marked. While this difference might be explained on the theory of an internal renal secretion, it would seem to us better regarded as the result of the difference in the urinary drainage going on persistently. In both groups there is persistent polyuria, but in one there is also persistent albuminuria, which is absent in the other; not unnaturally, the defective growth is more marked in the type with albuminuria.

The other symptoms, the changes in the urine and the peculiar family histories, are proper to diabetes insipidus, and require no special mention here.

Conclusions.

When further cases have been investigated they may show variations from the types we have here sketched. In Group 1 may be expected cases of cystic kidney or inherited syphilis, and in Group 2 instances of marked pituitary or syphilitic symptoms. From the cases we have studied, however, we may submit the following conclusions:

(1) A class of symptomatic infantilism secondary to a perversion of renal functions may be recognised ("renal infantilism").

(2) It may occur with organic renal disease; in the cases hitherto reported this type has been due to non-syphilitic chronic interstitial nephritis, whether cardio-vascular changes be present or absent. Here the infantilism is likely to be of a severe grade, and death tends to occur during childhood or early adolescence from uræmia or pneumonia.

(3) It may also occur apart from organic renal disease (diabetes insipidus). This type may be due to inherited syphilis, organic nervous lesions, and the other recognised causes of diabetes insipidus. The infantilism is not of a severe grade, and life may be prolonged.

(4) In either type the symptoms, polydipsia, polyuria and retarded development, may be present from birth or may develop during early childhood.

(5) Hitherto no case of either type has been materially affected by treatment.

References.

Group 1.

(1) Fletcher.—'Proc. Roy. Soc. Med.' (Child. Sect.), 1911, iv, p. 95.

(2) May.—'Univ. Coll. Hosp. Mag.,' 1912, ii, p. 99.

(3) Miller.—'Proc. Roy. Soc. Med.' (Child. Sect.), 1911, v, p. 38.

(4) Naish.—*Ibid.* (Child. Sect.), 1911, v, p. 41; 'Brit. Journ. Child. Dis.,' 1912, ix, p. 337.

(5) Parsons.—'Brit. Med. Journ.,' 1911, ii, p. 481.

(6) Sawyer.—'St. Thomas's Hosp. Rep.,' 1906, xxxv, p. 459.

Group 2.

(7) CHERRY.—'Austral. Med. Gaz.,' 1910, xxix, p. 302.

(8) FUTCHER.—'Trans. Assoc. Amer. Phys.,' 1904, xix, p. 247.

(9) PECHKRANC.—'Medycyna i Kron. lek. Warszawa,' 1911, xlvi, pp. 458, 482.

(10) WEBER.—'Brit. Journ. Child. Dis.,' 1912, ix, p. 211.

December 29th, 1913.

ORTHOPÆDICS IN MEDICAL PRACTICE. By Prof. ADOLF LORENZ and Dr. ALFRED SAXL. Authorised translation from German. By L. C. PEEL RITCHIE, M.D., F.R.C.S.Ed. Pp. 288, 39 illustrations. London: John Bale, Sons & Danielsson, Ltd. 7*s.* 6*d.* net.

For translating this work Dr. Ritchie merits the lively thanks of the profession. There exists without doubt a more intimate connection between internal medicine and orthopædic surgery than at the first glanc might eappear to be the case. To detect threatening mischief in its early stages and to submit it as soon as possible to appropriate specialist treatment is an important mission of the general practitioner, so that even a superficial knowledge of the more common orthopædic conditions may well emphasize the importance of the subject in medical practice. Some pitfalls are indicated, such as the omission to recognise psoas abscess, early disease of hip-joint treated for sciatica or the knee-joint treated instead of the hip. So also attention is drawn to the "pain in the belly" now and again complained of by children caused by a tuberculous spondylitis, and the vertebral inflammation mistaken for colic and treated by opium or some other symptomatic remedy. The bulk of the book is naturally occupied by treatment for diseases of the nervous system, but the digestive, locomotory, respiratory and circulatory systems are also discussed. The book will supply a felt want in general practice and may profitably be consulted, especially in children's diseases.

A CLINICAL LECTURE

ON

SOME OF THE CEREBRAL MANIFESTATIONS OF INFLUENZA.*

By FRANK E. TYLECOTE, M.D., D.P.H., M.R.C.P.,

Senior Physician to the Hospital; Visiting Physician to the South Manchester Union Hospitals.

GENTLEMEN,—In choosing my subject for this afternoon I have been influenced by the fact that we have recently been passing through an epidemic of influenza, which has shown itself in some of its less usual forms, and I therefore thought it might be useful to direct your attention for a short time to some cases in which this disease has manifested itself mainly by cerebral symptoms or in which cerebral complications have occurred.

We must at the outset divide these cases into three main groups, namely, those in which the cerebral symptoms date from the onset of the illness, those in which they have supervened a few days later, and lastly, those in which they have come on in the period of convalescence.

In the early stage of the ordinary catarrhal type of influenza severe frontal headache and pain on pressing the eyeballs or referred to them by the patient are common enough; less common symptoms of onset are attacks of giddiness and sudden unconsciousness. I have seen several cases admitted to hospital in a state of almost complete coma, with furred tongue, a slight or no rise of temperature, and not much alteration in the pulse-rate. Some of the patients, though they are unable to talk, appear to understand what you are saying. Here we are dealing with a profound toxæmia, and as a rule after two or three days the patient begins to improve, though it may be anything up to three weeks before he is mentally himself again. You will gather from this that I look upon these cases as having a favourable prognosis, and that is true if you limit it to those cases which are quiet and to those which show improvement in three days; those who are restless and actively delirious do not

* Delivered in the Post-Graduate Course at the Salford Royal Hospital.

do so well, and occasionally a case—usually an elderly patient—remains semi-comatose for a time; then the coma becomes deeper, and changes in the reflexes which one associates with a lesion of the upper neuron appear. In one such case, an old outdoor labourer of over seventy, a diagnosis of complicating cerebral thrombosis was made, and this was borne out by post-mortem three months later.

A year or two ago I saw a man, æt. 47 years, who had started with the usual pains in the head and back and feeling of lassitude. He, however, insisted on sticking to his work and refused to go to bed till three days later, by which time he had a severe bronchitis. It was three days later still when he first called in a doctor, who carefully examined him but found no pneumonia or other complication. The same night the doctor received an urgent call to see him again and found him with delusions of persecution and regarding religious matters, and trying to get out of the window. When he had calmed down somewhat he said that he felt he was going to die and seemed uneasy. The bronchitis was much as before. Now, for the first time, the temperature began to rise and gradually climbed till it reached 103° F. The delusions persisted, he became very difficult to manage, and the chest signs more marked, though no pneumonia developed. He died on the twelfth day. When I first saw him on the sixth day, except for the delusions and for his own idea that he was going to die, I should have given a good prognosis, but mental symptoms at this stage if without any signs of pneumonia are invariably of evil omen, and I never ignore a feeling of impending dissolution when it is expressed by a patient of usually stable mentality. The very toxic cases frequently have a cloud of albumen in the urine, but in this case in the specimen I examined there was none, though there was a heavy deposit of urates. A post-mortem, which was refused, would doubtless have shown multiple capillary cortical hæmorrhages—a hæmorrhagic encephalitis.

Another case was the man who is now before you. He is fifty years of age. His doctor sent him to my out-patient room with a note saying that the commencement of his illness had been an attack of abdominal influenza. But he sent him to me for a change in his speech, which had become somewhat slurring, like that of an early general paralytic, and he also had partial aphasia. He refused to come inside, so I gave him a quinine mixture, and impressed upon him that he should go straight to bed when he got home. A week later he came back, feeling stronger, but with his speech still affected. I continued the treatment, and added five-grain doses of iodide. There was no suspicion of syphilis in the case at all, and the history was quite negative in that regard. Now you see him before you a month later quite recovered—speech and reflexes in every way normal.

The patient I show you next is a man, æt. 35 years, who was admitted with a multiple neuritis following a typical attack of catarrhal influenza. Besides great weakness of the legs, with absent knee-jerks and much wasting, there was a right facial paralysis of Bell's type, and also some optic neuritis on the left side. He now walks moderately well, and the facial paralysis has quite gone. The optic neuritis is still present, though not so marked as it was. Here, then, is an example of nerve manifestations following an attack; in this case there was an interval of about a fortnight between the commencement of the attack and the onset of the peripheral neuritis. I believe, in these cases of motor neuritis, the same is true as of diphtherial neuritis, that, given proper treatment, if recovery occurs it will be absolute, *i.e.* without any permanent paralysis. I cannot recall ever having seen influenzal neuritis end fatally from involvement of either diaphragm or cardiac nerves as in diphtheria.

Very distressing, however, are the cases in which complete loss of taste or smell occurs early in the course of influenza, and is permanent. I have at present under my care a female patient, æt. 45 years, who complains that since an attack of influenza six years ago she has had no sense of smell, and "can only taste mustard at the back of her tongue."

Of the influenza patients who are delirious within a short time of the onset we have two main groups: firstly, the type where the delirium is due to the pyrexia, which in such a case is considerable. In this type the prognosis is better than in the second, in which the delirium is a sign of profound toxæmia, and where there is often little or no pyrexia—at any rate the delirium bears no relation to it. Here the prognosis may often be very bad.

Meningeal symptoms occurring at the onset of influenza or in its course are very interesting. In some cases, though there may be rigidity of the neck muscles and retraction of the head, there is no change in the fluid removed by lumbar puncture, and in a few days the symptoms pass off. These are cases of meningism or pseudo-meningitis, which may occur in the acute stages of several of the fevers, as I pointed out in a paper in the 'Medical Chronicle' some years ago, which you will find fully abstracted in the article on this subject in the last edition of Allbutt and Rolleston's 'System.' The condition is due to the circulation of toxins in the blood-stream.

In cases of true influenzal meningitis meningeal symptoms generally appear after a few days of catarrhal symptoms. Here there are definite changes in the cerebro-spinal fluid, and organisms are readily found. We sometimes find the influenza bacillus itself, sometimes the pneumococcus or *Micrococcus catarrhalis*, which latter two organisms are responsible for many epidemics of influenza, or it may be an infection of a secondary nature by some other organism, either one of the pus organisms or the tubercle bacillus, which last is, of course, very hard to find in the films. The prognosis in these cases naturally varies with the organism responsible. Many of the true influenzal cases recover. Some die, and in a few of these there is also encephalitis. Pneumococcal cases often recover in adults, but hardly ever in young infants, in whom they are often fatal in less than twenty-four hours.

Hemiplegia, monoplegia, and even paraplegia may follow influenza. The cause is variable; we may be dealing with a lesion of either upper or lower neuron. If the former, the cause may be a gross hæmorrhage in the course of a hæmorrhagic encephalitis, in which case the onset is usually early in the attack; if it comes on later it is often due to cardiac dilatation and thrombosis, with detachment of some of the thrombus, forming an embolus which is carried to the region of the internal capsule. If a lesion of the lower neuron it may be an acute anterior poliomyelitis.

Before we mention briefly certain of the mental troubles of the influenzal convalescent, I must remind you of those cases where at the height of the attack acute pain in the region affected heralds the onset of an acute catarrhal otitis media or of a frontal sinusitis, or of involvement of the maxillary antrum. The onset of this group of complications is often very sudden. These often do very well, but pus formation and bone necrosis may follow, and repeated operations be necessary.

Other complaints of the post-influenzal patient include vertigo, which may persist for many months. In some it only occurs when the patient looks down. Others have intractable neuralgia, in which case I have known cold compresses and blisters to give great relief when drugs have failed. As for changes in the mental state, cases of depression are more common than those of excitation. They are also of better prognosis, though their recovery may be a very long drawn out one. Some years ago I saw a lady who had had a bad attack of influenza, and the marked feature of it was that almost from the start she turned against certain of her relatives of whom she had previously been very fond; she imagined that she was suffering all manner of persecution from them. For six months these ideas remained as fixed as ever, and then, just as we were beginning to think they were permanent, they began to fade, and in two years from the commencement her mind was quite normal again. In other cases the condition may be merely one of extreme depression without any delusions; here, again, it may be months before there is any improvement, and to hurry it on we may suggest golf, a motor-car tour, or a voyage, always, however, remembering, if we order the last, the tendency to suicide which some of these cases may exhibit without the slightest warning or suggestion. Frequently the fear of such a catastrophe is too great to allow us to advise a voyage, but one of the other two suggested lines of treatment will prove successful in very many cases. At any rate a complete change will be necessary before any good can be done. And it is always well to remember that a patient who has once had a mental complication after an attack of influenza is more liable to similar complications with repeated attacks.

Insomnia after influenza may give us much trouble. As a general rule treat it, then, by plenty of good food and change of air rather than by drugs. Sometimes it is due to cerebral anæmia, at others it is from an accumulation of toxic products. If you must use drugs to give the patient a start, then I prefer the bromide group, and especially those compounds into which the urea radicle enters.

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